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Priestley Smith

Glaucoma

The Bowman Lecture

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CHAPTER VI.

GLAUCOMA.

By PRIESTLEY SMITH

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THE morbid condition known as glaucoma is one of the most serious to which the eye is liable. At one time gradual and insidious in its onset, at another sudden and violent, it is in all cases a progressive disease which, if neglected or incorrectly treated, leads sooner or later to incurable blindness. If, however, it be appropriately dealt with in its early stage, it may very generally be cured or arrested. It is therefore of great importance that the nature of the malady, and the particular measures by which alone its disastrous consequences can be avoided, should be clearly and generally understood.

It will be well to preface the clinical description of the disease by defining the present application of the term glaucoma, by briefly setting forth its essential characters, and by discussing the intricate question of its pathology. This done, the practical study of its symptoms and treatment will be more profitably undertaken.

The essential characteristic of glaucoma is an *increased tension of the eyeball.* The morbid process differs fundamentally from every other which attacks the organ of vision. It is not an inflammation, although it is often associated with inflammatory states; it is not an atrophy or a degeneration, although atrophic changes constantly attend its course; it is a peculiar and complex condition, depending for its existence upon an abnormal increase of the intra-ocular pressure. The fluid contents of the globe become increased in quantity by obstruction of the channels through which they normally escape; the tunics, being subjected to an increased pressure from within, are thereby put more tightly on the stretch; tested by the external pressure of the finger, the sclera is found to be unduly resistant, or, in other words, the eye feels harder than in health.

More serious than the increased tension of the envelope of the eye is the mischief which the pressure works upon the delicate structures within. The mechanism of the accommodation is impaired or paralyzed, and

the eye loses the power of altering its focus for different distances; the nutrition of the retina is impeded, and a corresponding loss of function follows; the nerves which perforate the sclera to traverse its inner surface are compressed and stretched, and suffer a marked diminution of conductility; the course of the blood which circulates in the retina and in the uveal tract is so much embarrassed that arterial interruption, venous turgescence, and sometimes capillary hæmorrhage ensue; the optic-nerve-entrance, the weakest point in the envelope of the eye, yields before the excess of pressure, and a cupped or excavated disc is the result. The imprisonment of the intra-ocular fluid gradually arrests the process of secretion, and the structures which depend upon this fluid for their nourishment—the lens and vitreous—degenerate; ultimately even the tough and resistant sclera yields to the continued pressure; it stretches and attenuates, with general or irregular enlargement of the eyeball, and finally may even rupture, and thus render the destructive process complete.

Glaucoma may occur alone, or as a complication of other diseases of the eye. In the former case it is termed *primary;* in the latter, *secondary* or *consecutive.* In their etiological relations the primary and secondary forms of the disease present considerable differences.

Primary Glaucoma is one of the less common maladies of the eye. Its frequency in this country, as computed from the published statistics of our large special hospitals, appears to be equivalent to one case of glaucoma in about 150 cases of eye-disorders of all kinds. Statistics from abroad show a somewhat higher proportion than this. It is pre-eminently a disease of advanced life, the large majority of all the cases met with being in persons over fifty years of age. It occurs occasionally, however, in young adults, and has been seen even in children; but such instances, especially the latter, are altogether exceptional. Women appear to be somewhat more liable to acute attacks of primary glaucoma than men, though in other respects the disease exhibits no preference for either sex. It appears more frequently in a chronic than in an acute form. A hereditary tendency has been distinctly traced in a considerable number of instances. The disease is met with in association with each of the three varieties of refraction, but it is considerably more frequent in hypermetropic than in emmetropic or myopic eyes.

Secondary Glaucoma, if we include within the term every case of ocular disorder which is complicated by an excess of tension, is met with more frequently than the primary disease; but the cases in which it is present are necessarily registered under so many different headings in our statistical records, that it is impossible to ascertain its frequency with any degree of precision. The ocular maladies which are especially liable to this complication are the following:—Serous iritis, posterior synechia when the whole of the margin of the pupil is implicated, anterior synechia when broad, injuries and dislocations of the lens, intra-ocular hæmorrhage, and intra-ocular tumour. Secondary glaucoma is often met with in association with staphyloma of the cornea and sclera, and with general enlargement of

the eyeball (*buphthalmos*). These latter conditions are to be regarded as consequences, and not as causes, of an excess of tension.

The glaucomatous condition is one which tends progressively to perpetuate and to intensify itself. It knows no *vis medicatrix Naturæ*. Treatment of an "expectant" nature, and measures directed against the subordinate symptoms, are alike useless—indeed, worse than useless, for they involve the loss of precious time.

The only real remedy for the disease is the reduction of the intra-ocular pressure.

The best means of effecting this reduction is usually the operation of iridectomy.

Prior to the year 1856 glaucoma was an incurable disease. Albrecht von Graefe was the first to fully recognise the significance of the increased tension of the eyeball, and to deliberately strive to find a means by which its reduction might be effected. The cure of glaucoma by iridectomy was the result of his labours. This discovery must ever be regarded as one of the most beneficent and brilliant achievements of our art; and it is of no little interest to note that although in von Graefe's day no satisfactory explanation of the tension-lowering power of iridectomy could be given, an advanced pathology now sanctions and confirms the procedure, even to the minutest details laid down by its author.

PATHOLOGY OF GLAUCOMA.

A study of the pathology of glaucoma must be based upon a knowledge of the normal intra-ocular pressure; we must know what is the amount of this pressure in the healthy eye, and what are the processes by which it is maintained, before we can analyse the causes of its pathological excess. A complete discussion of this subject would fill very many pages, and be out of place in this work. The salient points only can be indicated here. Should the reader desire to pursue the matter in detail, he may find in my previous essay on glaucoma* references to various original investigations, including some experiments of my own.

In certain of the lower animals the intra-ocular pressure has been measured by means of the manometer; in man it has been computed indirectly by the help of the tonometer, an instrument designed for the accurate measurement of the tension of the sclera. In man and animals alike it is found to be equal, approximately, to the pressure of 25 millimetres (1 inch) of mercury. The intra-ocular pressure, though much lower than the blood pressure, is intimately dependent upon this latter, and varies with its variations, the changes in the two cases being similar in kind, but different in degree.

In the healthy eye the pressure is equal in the aqueous and vitreous chambers, or if any inequality exist it is so slight as to be undiscoverable by

* "Glaucoma: its Causes, Symptoms, Pathology, and Treatment." (The Jacksonian Essay for 1878.) J. & A. Churchill. London, 1879.

any means at our command. It has been taught on high authority, and accepted by many, that the vitreous pressure is considerably higher than the aqueous pressure, but the doctrine appears to be no longer tenable. No excess of vitreous-pressure over aqueous-pressure can exist, unless the partition which separates the two chambers is capable of supporting the excess without displacement forwards, and this is not the case. Experiments of my own have proved that an extremely slight preponderance of pressure in the vitreous chamber overcomes the resistance of the lens and suspensory ligament, and drives them forward in advance of their normal situation. The only agent, then, by which the alleged excess could be neutralised is the iris; but it is certain that the iris plays no such part, for when it is completely paralyzed the lens does not advance. It may, therefore, be safely asserted that in the healthy eye the pressures which fall upon the lens and its suspensory ligament from in front and from behind are practically equal.

The maintenance of the intra-ocular pressure at its normal height demands a continuous balance between the secretion and the removal of the intra-ocular fluid. Whence comes this fluid? What purposes does it serve? Whither does it go?

The intra-ocular fluid is secreted chiefly, if not entirely, by the ciliary processes; it nourishes the vitreous body and the lens; it fills the space which intervenes between the lens and the cornea, constituting a medium in which the movements of the iris can be performed with the least possible amount of resistance; it escapes from the eye at the angle of the anterior chamber by filtering through the ligamentum pectinatum into Schlemm's canal and the veins in connection therewith. It has been supposed that the vitreous body is nourished by a fluid which is secreted by the choroid, and which reaches it by percolating through the retina; but there is no evidence in favour of this supposition, while there is much which tells against it. It is inherently improbable that a highly complex structure like the retina, consisting of many differentiated layers, and possessing an isolated vascular system of its own, should transmit the secretion of a distinct vascular tract situated on its one side to a structure with which it has nothing in common, either structurally or by development, situated on its other side. Moreover, recent discoveries connected with the so-called "visual purple" tend to show that the function of the chorio-capillaris and hexagonal epithelium is to regenerate the photo-chemical processes upon which retinal sensibility depends. And again, as showing that it is by the ciliary processes, and not by the uveal tract as a whole, that the intra-ocular fluid is secreted, we find that the choroid may reach a state of advanced degeneration, and that the iris may be atrophied or even absent, without any discoverable reduction in the fluid contents of the globe; whereas wasting of the ciliary processes appears to be invariably associated with a reduced supply of the intra-ocular fluid, leading in most cases to a shrinking of the vitreous body and detachment of the retina. And in confirmation of this view we find that the ciliary processes are

very specially adapted, both by structure and position, for this secretory function; their surfaces, extended by a series of intricate convolutions, are in relation posteriorly with the vitreous body, being separated from it only by the suspensory ligament, and in front of this they are free in the posterior aqueous chamber. Finally the secretory function of these organs has been amply demonstrated by injection experiments, especially, of late, by a new and very delicate method—the injection of fluorescine.*

The fluid secreted by the ciliary processes passes directly into the vitreous and aqueous chambers, and a portion enters the periphery of the lens. The waste fluid from the vitreous body passes forwards again through the suspensory ligament, and, together with that returning from the lens, mingles with the fluid which fills the posterior aqueous chamber. From the posterior chamber the fluid passes forwards through the pupil to gain the anterior chamber, and thence it escapes through the ligamentum pectinatum as already stated.

It has been suggested that a portion, at least, of the waste fluid of the vitreous body escapes through channels which are believed to connect the vitreous chamber with the subvaginal space of the optic nerve. If such channels actually exist (my own experiments lead me to doubt their reality), it is certain that they exercise no important influence on the tension of the eye; for artificial occlusion of every possible channel situated in the region of the optic nerve causes no retardation of the escape of fluid from the eye;† and, more significant still, dropsy of the nerve-sheath in conditions of exalted intra-cranial pressure induces no discoverable change in the internal pressure of the eye. On the other hand, the passage of fluid from the vitreous to the aqueous chamber has been positively demonstrated.

The fact that the intra-ocular fluid escapes from the eye at the angle of the anterior chamber, and does not exude through the cornea as was formerly supposed, we owe to a series of admirable experiments by Leber.‡ Coloured fluid injected into the anterior chamber rapidly appears in the episcleral vessels in the ciliary region. The path of the fluid from the angle of the chamber to the superficial vessels may readily be traced in microscopic sections from eyes in which this experiment has been performed. A confirmation of Leber's discovery is found in many facts connected with the pathology of glaucoma.

The physiology of the intra-ocular fluid, as it bears on the pathology of glaucoma, may be summarised as follows:—

The ciliary processes secrete a fluid which nourishes the vitreous body and the lens, and fills the aqueous chamber.

The fluid which returns from the vitreous chamber passes through the suspensory ligament into the posterior aqueous chamber. From the posterior aqueous chamber the fluid passes forward through the pupil into the anterior

* SCHOELER and UHTHOFF. *Vide* "Ophth. Review," Vol. I., p. 413.
† Experiments by SCHOELER. *Vide* "R. L. O. H. Rep." X., p. 28.
‡ "A. f. O." 19, II., p. 87.

chamber. It escapes from the interior of the eye at the angle of the anterior chamber.

The pressure of the fluid is practically equal in the vitreous and aqueous chambers, and is equivalent to about 25 millimetres of mercury.

The maintenance of this pressure demands a normal secretion and a normal excretion of the fluid.

An excess of the intra-ocular pressure must be due either to an accelerated secretion, or to a retarded excretion of the intra-ocular fluid; and it is certain that either one of these conditions is capable of inducing it.

Thus, compression of the abdominal aorta in animals, by increasing the amount and the pressure of the blood flowing to the head, exalts the tension of the eye; occlusion of the veins through which the blood escapes from the eye produces the same effect in smaller degree; and artificial irritation of the fifth nerve has been found to induce a very marked rise of the intra-ocular pressure, in association with a dilatation and pulsation of the arteries throughout the region of its distribution. In these cases the increased afflux of blood is doubtless associated with an accelerated secretion of the intra-ocular fluid. And on the other hand the discoveries of the last few years have shown that structural changes, such as must obstruct the escape of the intra-ocular fluid, are constantly to be found in association with an exalted tension.

To which class of causes, then, is the disease glaucoma to be ascribed? Is it the result of an accelerated secretion, or of a retarded excretion? The respective responsibility of these two agents appears to be as follows:—*Accelerated secretion is one of the exciting causes of glaucoma, but retarded excretion is the condition upon which the disease essentially depends.*

In the year 1876 Max Knies published a series of observations showing that, in eyes which have been blinded by glaucoma, the angle of the anterior chamber is commonly closed by adhesion of the periphery of the iris to the opposing surface of the cornea. Almost at the same time, Adolph Weber called attention, in like manner, to this same obstructive change. Taken in conjunction with Leber's demonstration of the excretory function of the angle of the anterior chamber, and with the fact that the operation of iridectomy involves an incision through the region in question, this discovery constituted a highly important step in the pathology of glaucoma, in that it gave a satisfactory physical explanation of the excess of tension. It did not, however, reveal the starting-point of the disease.

The mode in which the closure of the angle of the chamber is brought about had still to be made clear. Moreover, before the new explanation of the excess of tension could be generally accepted, it was necessary to account for certain cases of glaucoma in which no closure of the angle of the chamber exists; and for certain other cases in which the closure exists without an excess of tension. Further investigation has tended to

remove these difficulties, and to confirm the essential importance of the obstructive changes. Let us examine the nature of these changes, and the manner of their occurrence in the several varieties of glaucoma.

PATHOLOGY OF PRIMARY GLAUCOMA.—*Predisposing Causes.*—Primary glaucoma is met with in association with various general disorders, and it is met with in persons who appear otherwise healthy; it cannot be regarded as a local manifestation of any particular constitutional disease. The one condition to which it bears a clear relationship is *age*, the large majority of all the cases met with being in persons who have passed their fiftieth year. With the advance of life the healthy crystalline lens increases continually in size; during the forty years between the ages of twenty-five and sixty-five it adds about one-third to its volume, and about one-tenth to its linear dimensions (*vide* Fig. 126). The structures which surround the lens

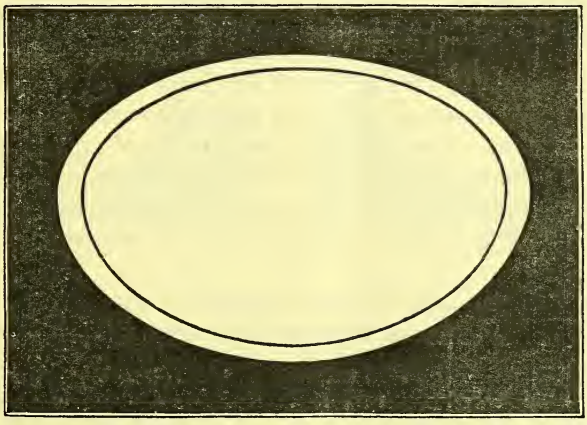

Fig. 126.*

DIAGRAM SHOWING THE RELATIVE SIZES OF THE AVERAGE LENS AT 25 AND 65 YEARS OF AGE.

attain their full size at the beginning of adult life if not earlier; the diameter of the cornea for example increases little if at all after seven or eight years of age.† Hence it comes about that as age advances the lens gradually encroaches upon the space in which it lies, compensation being obtained at the expense of the aqueous chamber; the anterior chamber becomes shallower, the *circumlental space*—the space which separates the margin of the lens from the ciliary processes—becomes narrower (*vide* Figs. 127 and 128). The change is a physiological one, and is entirely compatible under all ordinary circumstances with the perfect integrity of the eye, but it increases the liability to disease of a certain kind—the obstructions in the path of the intra-ocular fluid which characterize glaucoma arise more easily where the channels are already

* From "Brit. Ophth. Society's Trans." Vol. III.

† EMMERT. "Auge und Schaedel," Berlin, 1880, p. 21.

narrow than where they are wide. It may, I think, be confidently asserted that *the special liability to primary glaucoma which belongs to the senile eye depends upon the large size of the senile lens.*

Hypermetropia appears to predispose in some degree to primary glaucoma. The explanation will probably be found in the subnormal dimensions and peculiar conformation of the hypermetropic eye; the anterior chamber is shallower, and the ciliary processes are more prominent in the direction of the lens than in the emmetropic or myopic eye.

It is said that the rigidity of the sclera proper to the senile eye is a strongly predisposing factor. The idea has, I think, no good foundation, for though primary glaucoma is rare in early life secondary glaucoma is common even in the highly elastic eyes of children; this shows that elasticity confers no immunity from glaucoma if the outlets of the eye become obstructed. Predisposition lies in a liability to obstructive changes, not in a loss of elasticity. The influence of the latter is manifest in the result rather than in the causation of the disease—a pressure which the senile sclera can withstand for years will stretch the youthful sclera to an excessive size in the course of a few months.

Exciting Causes.—The immediate local cause of primary glaucoma is pressure of the ciliary processes against the base of the iris and consequent compression of the angle of the anterior chamber; it may arise from any disturbance which brings the processes and the margin of the lens into undue proximity.

In many cases the fault seems to lie chiefly in the processes; by reason of some vascular disturbance they swell to more than their usual size; the circumlental space being already of small dimensions is abolished; the processes expand in the only direction which is open to them, namely, forwards, and in so doing push the base of the iris forwards against the cornea. These changes are strikingly apparent in specimens of acute glaucoma excised early in the course of the disease.

In other cases the fault lies very probably in the lens. The growth of the lens is on the average very uniform throughout life, but at every age there are individual lenses of very different sizes.* It is likely that in some cases of simple chronic glaucoma in which all signs of vascular disturbance are wanting the lens is of excessive size. Or if it be not absolutely large as compared with the average lens, it may be relatively too large for the eye to which it belongs. It is almost impossible to determine this point by observation, for measurements made after the disease has run its course do not represent with certainty the dimensions at an earlier period. When the circulation of the intra-ocular fluid is arrested, nutrition ceases, and when nutrition ceases shrinking commonly begins. It is asserted that lenses removed from glaucomatous eyes after excision are on the average somewhat below the normal size.†

* *Vide* Chart.—"Brit. Ophth. Society's Trans." Vol. III.

† W. A. Brailey, "R. L. O. H. Rep." X., p. 372.

No sooner does the compression of the angle of the anterior chamber begin, than a "vicious circle" is created in which the mischief tends to become self-intensifying. The increasing pressure within the eye, falling upon the choroidal veins throughout the whole of their course, embarrasses the blood-stream in them, and sets up a passive hyperæmia in the ciliary processes, evidence of which is seen in the compensatory dilatation of the anterior ciliary veins. The overloaded processes in striving to expand, intensify the blockade; on the one hand they come into still closer and wider relation with the margin of the lens, while on the other they compress the angle of the anterior chamber still more firmly (Fig. 129).

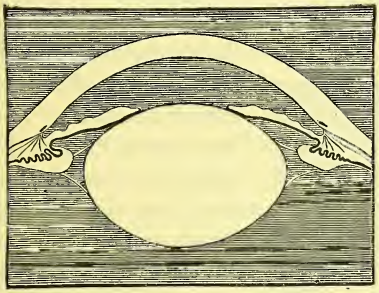

Fig. 127.

From a Healthy Eye, at Age 21.

Showing angle of anterior chamber open opposite Schlemm's canal; lens comparatively small; circumlental space wide. (*Enlarged* $3\frac{1}{3}$ *diameters.*)

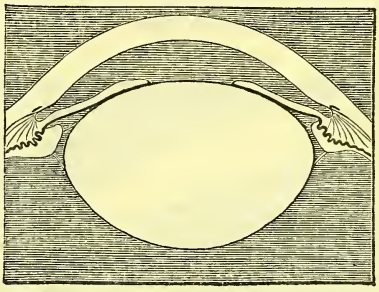

Fig. 128.

From a Healthy Eye, at Age 90.

Showing angle of anterior chamber open; lens comparatively large; circumlental space narrow. (*Enlarged* $3\frac{1}{3}$ *diameters.*)

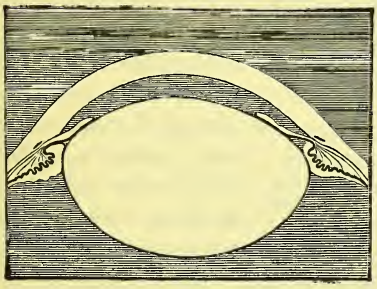

Fig. 129.

From a Specimen of recent Glaucoma, at Age 66.

Showing margin of lens in contact with ciliary processes; angle of anterior chamber closed by pressure of processes against iris. (*Enlarged* $3\frac{1}{3}$ *diameters.*)

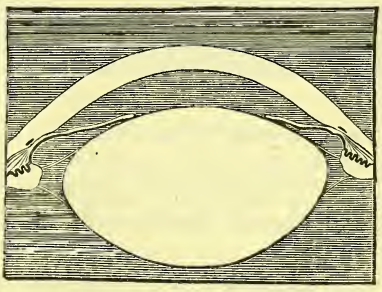

Fig. 130.

From a Specimen of Glaucoma of Long Duration, at Age 69.

Showing angle of anterior chamber closed, as in Fig. 3; ciliary muscle and ciliary processes atrophied and displaced outwards by long-continued pressure; lens of unusually large size. (*Enlarged* $3\frac{1}{3}$ *diameters.*)

Note.—All the figures in this chapter, with the exception of Nos. 134, 135, 140, 141, 142, were drawn from actual specimens by means of the microscopic camera, and reduced by photography upon the wood block. *Vide* "Ophth. Rev." Vol. II. p. 69.

Simultaneously with the occurrence of these obstructions the anterior chamber usually decreases somewhat in depth, and in some cases becomes extremely shallow. This is, at first sight, difficult to reconcile with the indubitable turgescence of the ciliary processes, for these organs lie *in front of the suspensory ligament*, and any increase of their dimensions must tend, *per se*, to displace the lens backwards, whereas what actually happens is that the lens advances and the processes are squeezed, sometimes into a remarkable wedge-like form, between it and the iris (*vide* Fig. 131). It is clear that the intra-ocular fluid collects in excessive quantity in the vitreous chamber and expands it at the expense of the aqueous chamber. The probable explanation of its doing so is to be found in the altered relations of the ciliary processes; posteriorly they are still free to unload themselves if they can into the vitreous body, but anteriorly a large portion of their secreting surface is tightly pressed between the lens and iris; and in addition to this, the outlet of the vitreous chamber, the circumlental space, is closed so that the surplus vitreous fluid can no longer pass forward into the aqueous chamber as in health.

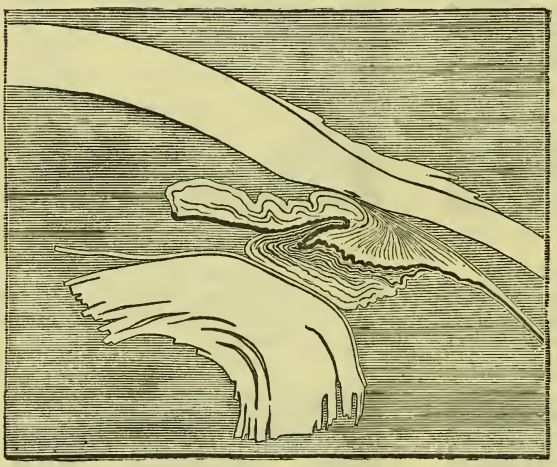

Fig. 131.

From a Specimen of recent Acute Glaucoma, at Age 74.

Showing margin of lens in contact with swollen ciliary processes; angle of anterior chamber closed by pressure of processes against iris. (The same condition exists at other parts of the circle. The lens was broken in dissection.) (*Enlarged* 10 *diameters.*)

In the *chronic*, non-irritative form of primary glaucoma the obstructive changes which have just been described come about very gradually. Clinically we see that there is little vascular disturbance, and that the relationship to senility is an almost invariable one. In this form of the disease it appears probable that the narrowing of the circumlental space depends rather upon a structural disproportion between the size of the lens and its surroundings, than upon any morbid turgescence of the processes (*vide* Note at end of Chapter, p. 652). The circumlental space is reduced by slow degrees, the ciliary processes undergo no considerable swelling, and, unless some auxiliary cause should supervene to induce hyperæmia and turgescence, a long period may elapse before the angle of the anterior chamber becomes completely closed and impermeable.

In *acute* glaucoma, on the other hand, the vascular disturbance is great. The outbreak of the attack commonly follows close upon some emotional,

neuralgic, or circulatory disturbance, such as would overfill the vessels of the head and eye. It is not necessarily preceded by glaucoma in a simple form; it requires for its occurrence only that the lens and the ciliary processes shall be already in dangerous proximity. Then a little local irritation, or a little cerebral hyperæmia, quickens the blood-stream to the eye, the ciliary processes swell up, the angle of the anterior chamber closes, and acute glaucoma is rapidly established. In this variety of primary glaucoma the obstructive changes are induced chiefly by the sudden swelling of the ciliary processes; at the same time it is obvious that the large size of the lens which is proper to the senile eye, is a predisposing factor of no less importance in the acute than in the chronic form of the disease.

In the early stages of the disease the angle of the anterior chamber is narrowed or closed simply by the pushing forward of the iris; in the later stages the surfaces of iris and cornea become firmly welded together by adhesive inflammation (*vide* Figs. 146 and 147, p. 636). We find such an adhesion in the great majority of eyes excised on account of advanced glaucoma. The rapidity with which it forms varies probably with the type of the disease, *i.e.*, in accordance with the amount of vascular disturbance. In a very small proportion of cases even of long standing no adhesion is found and the evidence of compression is comparatively slight. I have met with no instance in which evidence of obstruction at the outlet was entirely wanting.

The mode in which the operation of iridectomy relieves the glaucomatous condition, the beneficial action of eserine, and the danger which attends the use of atropine, are matters which bear closely upon the foregoing explanation of the pathology of the disease; but it will be more convenient to discuss them in a later section, in connection with the description of its treatment.

The theory contained in the foregoing paragraphs was put forward in my previous work in a somewhat different form, as a matter of inference, and as a basis for further investigation. It has been tested and modified by further research, and the evidence upon which it now rests is, I think, very strong; nevertheless, I would beg the reader to observe that the whole question of the dependence of glaucoma upon obstructive changes is a recent one, and that the explanation here given of the primary form of the disease is not as yet generally accepted.

It will be well to indicate in a few lines the mode in which this theory of glaucoma was arrived at, and the evidence on which it rests. Theodore Leber gave a new direction to the pathological inquiry by demonstrating the excretory function of the angle of the anterior chamber. Max Knies and Adolph Weber, independently, discovered the closure of this outlet in the glaucomatous eye. Weber showed, moreover, that the closure is caused by the pressure of the ciliary processes against the iris. Experiments of my own revealed the fact that a very slight excess of pressure in the vitreous chamber causes the lens and its suspensory ligament to advance, and drives the ciliary processes forward, so as to close the angle of the chamber in a manner closely simulating the conditions which exist in primary glaucoma. From this it was inferred that the starting-point of the

malady is some change which induces a slight preponderance of vitreous over aqueous pressure. It appeared to be a reasonable hypothesis that the change might be found in an obstruction situated at the outlet of the vitreous chamber—for example, a narrowing of the circumlental space. The following considerations, amongst others, favoured the idea that a diminished space between the lens and the ciliary processes is actually the starting point of primary glaucoma :—The disease occurs almost exclusively during the second half of life, when, as I presumed, the diameter of the lens is greatest, and the circumlental space narrowest ; injuries of the lens which cause it to swell as a whole are very frequently followed by glaucoma, and more frequently so in adults, in whom the lens is large, than in children, in whom it is small ; swelling of the ciliary processes is undoubtedly a part of the glaucomatous attack, and this also would tend to narrow or to close the circumlental space.

The theory thus arrived at afforded definite indications for further inquiry. It demanded further information as to the size of the lens at different periods of life, and as to the patency of the circumlental space in youthful, in senile, and in glaucomatous eyes. Ten healthy eyes (five pairs) from subjects aged 21, 26, 49, 65, and 90 were examined with special precautions as to non-disturbance of parts, and showed a progressive increase in the diameter of the lens with age, and a corresponding diminution of the circumlental space ; they showed, also, what I had not anticipated, a progressive increase in the antero-posterior diameter of the lens as well.* On subsequently examining a much larger number of lenses by a method of increased accuracy, I ascertained it to be a general rule that the lens continues to increase in volume and in weight throughout life.† Finally, there is direct evidence to connect this fact with the occurrence of glaucoma. In specimens of recent acute primary glaucoma I have found the ciliary processes swollen, and in close and forcible contact with the margin of the lens on the one hand, and with the periphery of the iris on the other ; the angle of the chamber being closed by the compression. It is only in recent cases that these primary obstructive changes can be found still existent ; atrophy of the ciliary processes and the subjacent muscle soon sets in, and with the shrinking and retraction of these structures the relation of the parts alters, and the circumlental space becomes ultimately even wider than in health (Fig. 130). A persistent excess of tension will widen the circumlental space to a very remarkable extent in the youthful eye by reason of the gradual stretching of the tunics (Fig. 132), and it doubtless operates in the same way in the more resistant eye of the adult, though in a lesser degree. Even in specimens of long-standing glaucoma, however, the base of the iris frequently bears an impression which marks the position occupied by the processes at the commencement of the disease, and which, taken together with the size and position of the lens, tells of a bygone closure of the circumlental space. Vestiges of previous contact are sometimes discoverable upon the margin of the lens by the microscope after excision, and by the ophthalmoscope after iridectomy.

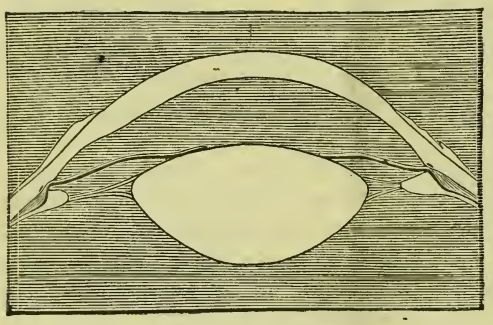

Fig. 132.

From a Specimen of Secondary Glaucoma, at Age 14, with great Enlargement of the Eye in all Diameters—Buphthalmos.

Showing angle of anterior chamber closed; cornea distended, and thinned towards the ciliary region; circumlental space greatly widened; ciliary muscle atrophied. (*Enlarged* 3½ *diameters.*)

* "R. L. O. H. Rep." x., p. 25. † "Brit. Ophth. Society's Trans." Vol. III.

Pathology of Secondary Glaucoma.—In many of the cases in which glaucoma arises secondarily to other diseases of the eye, the complication is due to a closure of the angle of the anterior chamber similar to that which occurs in primary glaucoma, though brought about in a somewhat different way; in other cases, an obstruction to the escape of the intra-ocular fluid arises from other causes, the outlet being choked by extravasated blood, or by fibrinous deposits from the fluid in the anterior chamber; occasionally a complete blockade of the anterior chamber is set up by impaction of the lens between the iris and the cornea. For a description of the diseases which lead to secondary glaucoma, the reader is referred to other chapters; here, only the mode in which the complication appears to be brought about will be indicated.

Posterior Synechia when extensive is a fruitful source of glaucomatous complication (Fig. 133). When the whole of the pupillary margin becomes adherent to the lens capsule, the intra-ocular fluid can no longer find its way forward through the pupil into the anterior chamber. It collects between the iris and the lens and suspensory ligament, and distends the posterior aqueous chamber. The iris is bulged forwards until, by its advance, it so far narrows the angle of the anterior chamber as to preclude the further escape of fluid into Schlemm's canal. The fluid still remaining in the anterior chamber is imprisoned there, and presents an unyielding resistance to any further advance of the iris, or the lens. Fluid is still secreted, though in decreasing quantity, into the vitreous chamber, and continues to collect there until its exalted pressure, reacting on the secreting organs, arrests the further supply, or reduces it to a condition of equilibrium with the small amount which still finds an escape from the eye. In recent cases of this kind the effect of iridectomy is most striking. No sooner is a communication re-established between the posterior and anterior aqueous chambers, than the disease—*i.e.*, the glaucomatous complication—vanishes. When, however, as in cases of long duration, the periphery of the iris has already formed a solid adhesion to the cornea, little or no benefit is derived from the operation. The glaucomatous tension, in cases of annular posterior synechia is apt, sooner or later, to be replaced by a subnormal tension indicating an inflammatory disorganization of the ciliary processes, and a consequent impoverishment of the vitreous body. In some cases iridectomy at once reduces the tension from a state of excess to one of persistent deficiency. Previous

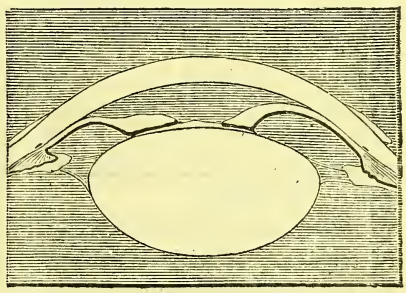

Fig. 133.

From a Specimen of Secondary Glaucoma induced by Annular Posterior Synechia.

Showing occlusion of pupil; iris pushed forward by fluid imprisoned in posterior aqueous chamber; angle of anterior chamber closed thereby. (*Enlarged* $3\frac{1}{8}$ *diameters.*)

to the operation in such a case the ciliary processes, although damaged, have secreted an amount of fluid which, by reason of the obstruction at the outlet, suffices to maintain the tunics in a state of high tension; but no sooner is this channel re-opened than the impoverishment of the supply becomes manifest.

When the whole of the posterior surface of the iris becomes cemented to the lens-capsule and suspensory ligament, the obstruction to the stream of the intra-ocular fluid is still more complete. The posterior aqueous chamber is abolished. Any secretion which may still issue from the ciliary processes is imprisoned in the vitreous chamber. The partition formed by the adherent structures advances until by so doing it closes the outlet of the anterior chamber, and locks in any aqueous fluid which may still remain within it.

Anterior Synechia, especially if a considerable portion of the circle of the iris be involved, readily leads to closure of the corresponding part of the angle of the anterior chamber. A perforating ulcer, or a wound of the cornea, is commonly the starting-point. The iris prolapses, and is included in the cicatrix; it is tightly stretched between its peripheral attachment and the new adhesion; the outlet of the chamber is compressed throughout the corresponding portion of the circle. If the obstructed portion is large enough to materially affect the escape of the intra-ocular fluid the tension of the eye rises. When such a condition is complicated by injury and swelling of the lens, with posterior adhesion of the iris to the capsule, the danger of glaucomatous complication is greatly increased.

Traumatic cataract is frequently associated with a considerable excess of tension. If iritic complications are set up by the injury, these may account for a closure of the angle of the anterior chamber in the manner already described; but in many cases a different explanation is necessary, for we see the most pronounced glaucoma rapidly induced by wounds of the lens—*e.g.*, needle operations—which leave the iris entirely uninjured. The agent in such cases is the swelling of the lens which accompanies a rapid imbibition of the aqueous humour; the resulting changes closely resemble those which have been assigned as the cause of primary glaucoma, namely, compression of the ciliary processes against the periphery of the iris, and closure of the angle of the anterior chamber. The liability to secondary glaucoma after injury of the lens is much greater in adults than in children, by reason, doubtless, of the larger size of the adult-lens. The prompt removal of the swollen lens-substance is usually followed by the immediate subsidence of the tension, If, however, the glaucomatous condition be allowed to go unrelieved too long, removal of the lens may fail to restore the patency of the angle of the anterior chamber, and the excess of tension may return with the closure of the wound, although the lens be gone.

Extraction of cataract is occasionally followed by an excess of tension in eyes in which no tendency to glaucoma has previously manifested

itself. It is probable that inflammation of the iris and the formation of adhesions with the capsule, or cornea, are usually the cause of the complication. In the only instance in which it has arisen in my own practice, the extraction of the lens was followed by acute iritis with copious plastic exudation, which almost hid the membrane from view, and blocked up the pupil by thickly coating the unruptured posterior capsule, and thus, by cementing these structures together, formed a thick impervious partition between the vitreous and aqueous chambers. This partition was ruptured by operation during the height of the inflammation in the hope of reducing the excessive tension of the globe, and thus saving the sight from otherwise certain destruction; the proceeding was justified by the result, for the eye recovered a normal tension, a clear pupil, and excellent vision. When the contents of the anterior chamber cleared up it became manifest that throughout a certain portion of the circle the periphery of the iris was adherent to the cornea. Whether the outlet was previously compressed throughout the whole circle by an advance of the iris, or whether the inflammatory material thrown off so copiously by the iris was the chief cause of the obstruction, cannot be determined. In all the examples of exalted tension following cataract extraction which have come under my own notice there have been iritic adhesions, but it is stated that the complication occurs occasionally even when the healing process has appeared to run a normal course. In such a case one would be inclined to suspect the presence of some hidden inflammatory changes in the tissues around the angle of the anterior chamber, or possibly of some deposits in this region from an intra-ocular fluid of morbid constitution, such as occur in serous iritis.

Dislocation of the lens into the anterior chamber is sometimes immediately followed by glaucoma of great severity. This sequel is not confined to instances of traumatic displacement; it may occur, also, when a lens which has been previously separated from its attachments passes forward through the pupil in consequence of a prone position of the face. The rise of tension is induced in a purely mechanical manner. Under normal conditions the intra-ocular fluid, in passing from the posterior to the anterior aqueous chamber, meets with no resistance at the pupil, the edge of which is raised from the surface of the lens to an imperceptible extent by the minute current; but no sooner is the relation of these structures reversed than the stream which previously kept the passage open tends to close it with constantly-increasing pressure, by applying the iris more and more firmly to the posterior surface of the lens. The intra-ocular fluid can now no longer enter the aqueous chamber. The periphery of the iris, where it is unsupported by the lens, is driven forward by the pressure from behind, so as to close the angle of the chamber. The degree of tension, the vascular injection, the pain, and the constitutional disturbance—*e.g.*, vomiting and nervous prostration—may be quite equal to those which accompany acute primary glaucoma, yet they subside with great rapidity when the obstruction is removed either by

extraction of the lens, or by its return to a position in the rear of the iris. In other instances of this same displacement there is an entire absence of the glaucomatous complication—no rise of tension, no injection, no pain. The difference is to be explained, I believe, by the varying sizes of the displaced lenses. If the lens be small or shrunken it will lie eccentrically in the chamber, so as to leave a free passage between its

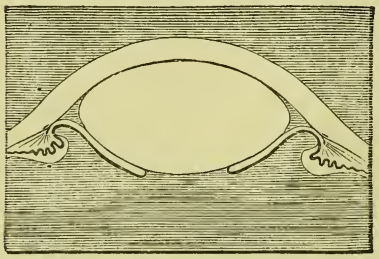

Fig. 134.

Dislocation of Lens into Anterior Chamber associated with Secondary Glaucoma (Ideal Representation).

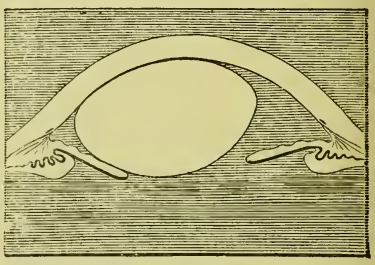

Fig. 135.

Dislocation of Lens into Anterior Chamber not associated with Secondary Glaucoma (Ideal Representation).

upper and hinder part and the margin of the pupil; if it be large enough to nearly fill the chamber, the iris is far more liable to become applied throughout to its posterior surface.*

Figs. 134 and 135 illustrate this explanation diagrammatically. Since they were drawn I have been able to examine a well marked example

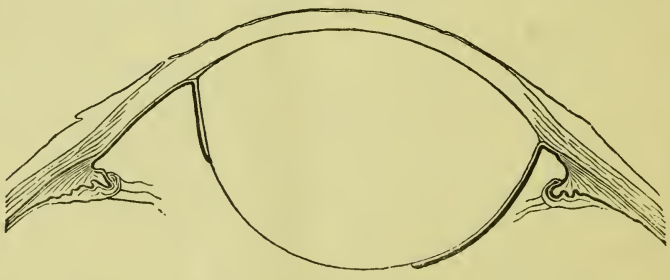

Fig. 136.

From a Specimen of Secondary Glaucoma induced by Dislocation of Lens into Anterior Chamber.

(*Enlarged* 5 *Diameters.*)

of this form of secondary glaucoma (Fig. 136). The eye was previously nearly blind from retinitis pigmentosa; the lens was displaced spontaneously; intense glaucoma followed at once; the anterior chamber was entirely abolished, the iris, where not supported by the lens, being closely

* *Vide* Cases, "Glaucoma: its Causes, etc." pp. 262-265.

applied to the cornea by pressure from behind. * One case has been placed on record in support of the explanation here given, in which no rise of tension occurred until, by the use of eserine, the iris was contracted and tightened up against the posterior surface of the lens, when an acute glaucomatous condition at once supervened. † The case is all the more noteworthy as a proof of the mechanical causation of the glaucoma, for eserine has a precisely opposite effect upon the tension where the relation of the parts concerned is different—it reduces the tension of primary glaucoma. Further, the case suggests the practical lesson that if eserine be employed as a preliminary to the removal of the lens from the anterior chamber, its use should be begun but a very short time before the operation.

Lateral dislocation of the lens by injury is not unfrequently followed by a rise of tension. This condition also I have lately been able to study both in the living eye and afterwards under the microscope. The angle

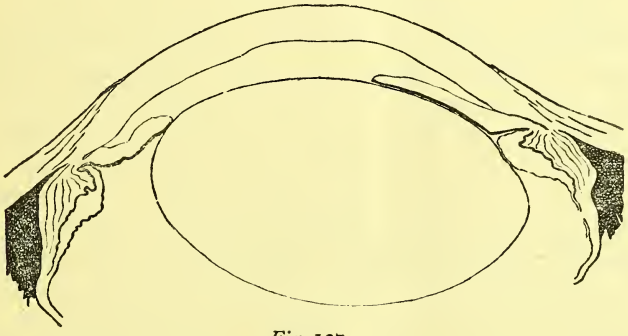

Fig. 137.

From a Specimen of Secondary Glaucoma induced by Lateral Dislocation of the Lens. (*Enlarged* 5 *Diameters.*)

of the chamber was firmly closed throughout that part of the circle towards which the lens was displaced, by the direct pressure of the margin of the lens against the ciliary processes (Fig. 137).‡ A glance at the normal relations of these parts, in the senile eye especially (*vide* Fig. 128), will show how readily such pressure may be set up by a slight tilting or lateral displacement of the lens. Several years ago I had under notice the case of an elderly man with traumatic lateral dislocation of the lens, in which the glaucomatous tension subsided as soon as the pupil was well dilated by atropine, and the detached margin of the lens freely exposed in the area of the pupil; returned in full intensity when the pupil again contracted; and again disappeared under the use of atropine. It seems not unlikely that the enlargement of the pupil permitted some slight alteration in the position of the lens which relieved

* "Ophth. Review," Vol. I., p. 209.
† J. J. Minor, "New York Medical Journal," 1881, p. 194.
‡ "Ophth. Review," Vol. II. p. 257.

the pressure of its hidden margin against the ciliary processes. In the other case referred to (Fig. 137), a single application of eserine reduced the tension to the normal, though the benefit was of short duration. It is worthy of note that the vitreous body, being an organised tissue and not a liquid, must offer a certain elastic resistance to the inroad of the displaced lens, and may not improbably tend to press the lens against the ciliary processes. Possibly it may itself be so displaced by the dislocated lens as to become the agent by which the pressure against the processes and the closure of the angle of the anterior chamber are effected. Thus, displacement of the lens backwards would drive the vitreous forwards round its margin, and would be likely, if the vitreous have a healthy consistency and cohesion, to drive the periphery of the iris forwards; while a disintegrated fluid vitreous would flow into the space vacated by the lens without creating local pressure. Clinically we see that a degenerated lens may fall back into a fluid vitreous without any tendency to excite glaucomatous complication.

A mechanical interference with the outlet of the anterior chamber, though certainly the usual and chief cause of high tension after dislocation of the lens, is probably not the only one; hæmorrhage, inflammatory changes in the ciliary region, or morbid alterations of the fluid poured into the chambers, may be concerned in producing it in some cases.

Tumours of the retina and choroid, at some stage of their growth, almost invariably induce a marked increase of the tension of the eyeball. The immediate cause of the glaucomatous condition is a closure of the angle of the anterior chamber produced by the pressure of the ciliary processes just as in primary glaucoma. The mechanism of the process is uncertain. It is important to note that the closure does not depend upon any direct pressure by the new growth upon the lens or ciliary processes, for it occurs when the anterior limit of the tumour is far removed from these structures. The intrusion of a foreign growth upon the space normally filled by the vitreous body, unless compensated for by an equivalent atrophy of the latter, must tend to overfill the vitreous chamber, and, as my own experiments have shown, a very small preponderance of vitreous pressure drives the lens forwards, and, through the agency of the ciliary processes, compresses the outlet of the anterior chamber; this is possibly the explanation of the cases in question. Again, we find in some of these cases a very pronounced turgescence of the ciliary processes; probably the increased afflux of blood to the uveal tract, supplemented possibly by compression of the veins in the neighbourhood of the tumour, tends to the production of this condition, and hence to glaucoma as in the primary form of the disease. Tumours of the choroid appear to secrete, or to excite the secretion of, a highly albuminous fluid, which, as the vitreous shrinks, is poured out between the choroid and retina, and ultimately causes the latter membrane to apply itself closely to the lens and suspensory ligament, these structures being driven forwards and the outlet of the anterior chamber compressed in the manner already described.

Tumours of the iris are less commonly productive of glaucoma than those which are seated more deeply, probably because they are more conspicuous, and therefore meet with surgical interference before they extend sufficiently to seriously obstruct the outlet of the anterior chamber. Through the kindness of Prof. McHardy I have been able to examine one striking exception to this rule (Fig. 138). The iris and ciliary processes were infiltrated throughout by a dense mass of small round cells, and were enormously increased in thickness; the circumlental space and the angle of the anterior chamber were completely blocked. The appearances presented a sort of exaggerated picture of acute primary glaucoma. The

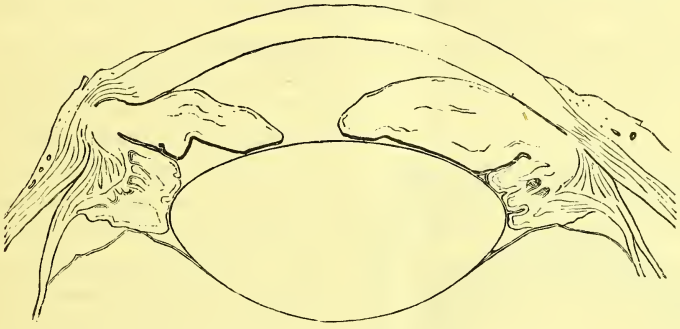

Fig. 138.

From a Specimen of Secondary Glaucoma induced by Tumour of the Iris. (*Enlarged* 5 *Diameters.*)

eye was stony hard when the patient came under notice, and there had been violent pain for several months. The disc was deeply cupped.

Tumours external to the eye occasionally induce glaucoma. It may be presumed that they do so by reason of the vascular disturbance which accompanies their growth—enlargement of afferent and compression of efferent vessels.

Intra-ocular hæmorrhage is not infrequently the immediate cause of a glaucomatous attack. Occasionally, perhaps, a retinal hæmorrhage may be regarded as an early symptom of a commencing glaucoma—that is to say, as a consequence rather than as a cause of increased tension; but in many cases, certainly, the glaucoma is truly secondary to the extravasation of blood. The sudden pouring out of blood into the interior of the eye must in all cases temporarily raise the intra-ocular pressure. Commonly the excess is rapidly removed by a compensatory escape of intra-ocular fluid; but if such compensation be difficult or impossible, the case is different. It sometimes happens that an eye which has been long disorganised and of subnormal tension is suddenly seized with violent pain, and becomes at the same time almost stony hard. After enucleation, dissection demonstrates an entire absence of the vitreous body, the retina detached and gathered together into a central bundle, which forms with

the lens, suspensory ligament, ciliary processes, and iris, a thick septum cemented together by old inflammatory exudation; and we find a quantity of blood poured out into the posterior part of the globe. In such an eye the secretory function of the ciliary processes has long ceased, and the channel which normally conducts the intra-ocular fluid from the vitreous chamber no longer exists; hence the non-removal of the effused blood, the absence of any rapid compensation for its presence, and the high tension. Prompt excision of the eye is advisable. Hæmorrhage into the anterior chamber will sometimes, in like manner, set up a high degree of tension. This is little likely to occur when an incision in the cornea, as after the operation of iridectomy, provides a channel for the speedy escape of the blood. It happens sometimes when the anterior chamber fills with blood as the result of an injury without penetration. It is more especially apt to ensue in eyes in which, from previous disease, the outlet of the chamber is already closed by compression, or impervious through long disuse. I have specimens of secondary glaucoma in which the angle of the anterior chamber is completely blocked with blood. Of more practical interest are those cases in which a deep-seated hæmorrhage in a previously sound eye is followed at once, or within a short space of time, by an attack which resembles acute primary glaucoma. When, by reason of the senile reduction of the circumlental space, an eye is already prone to become glaucomatous on the occurrence of even a small rise of vitreous pressure, it is conceivable that the mere addition of a small quantity of blood to the contents of the vitreous chamber might start the malady. In some instances, however, the extravasated blood acts in a more direct fashion; for in two of my specimens it has blockaded the circumlental space by entering and passing round the canal of Petit, thus establishing an obstruction similar in its effect to that which has been assigned as the starting-point of primary glaucoma, but one which, unlike a simple narrowing of the circumlental space from swelling of the processes, would not be relieved by iridectomy.

Serous iritis is usually associated with an exalted tension. This form of glaucoma differs from almost every other in that the anterior chamber deepens as the pressure rises, showing that the surplus fluid is collecting in the aqueous and not in the vitreous chamber. The retrocession of the iris renders it unlikely that any compression of its periphery is present during the glaucomatous attack, and in a specimen of serous iritis in my possession the angle of the anterior chamber is not only uncompressed, but distended far more widely than in the healthy eye (Fig. 139). For this specimen also I am indebted to Prof. McHardy. It is probable that the morbid constitution of the aqueous humour is the source of a retarded filtration in these cases. Serous iritis is characterized by the deposit of numerous small coagulated masses upon the posterior surface of the cornea, which, when very abundant, lead to parenchymatous inflammation and sclerosis of the corneal tissue itself. They settle by gravitation chiefly upon the lower half of the cornea. It is highly probable that

similar coagulations are deposited in the fibrous network through which the morbid fluid must filter in order to escape from the angle of the chamber, and that an impediment sufficient to cause an excess of tension is thus created. It is likely, also, that the highly albuminous nature of the fluid is of itself an impediment to the filtration process. In the specimen above mentioned there is a very manifest alteration, probably from fibrinous deposits, in the tissue immediately lining the angle of the anterior chamber, and throughout the lower half of the circle the angle contains a considerable quantity of free deposit or sediment.

Sympathetic inflammation, induced by morbid conditions of the fellow-eye, is sometimes associated with an excess of tension. When, as is commonly the case, the sympathetic affection appears in the form of a plastic iritis, with rapid formation of posterior adhesions, the occurrence of secondary glaucoma is readily to be understood (*vide* p. 609). Occasionally, however, a condition more nearly resembling an attack of primary glaucoma appears to be induced by sympathetic influence. The case is recorded of a female, aged forty-nine, in whom attacks of pain in an eye blinded by injury were accompanied on each occasion by redness, pain, and dimness, in the fellow-eye, and ultimately by well-marked acute glaucoma, which was cured by iridectomy. I have lately seen a case in which, after the subsidence of symptoms of sympathetic irritation, the tension was high, the anterior chamber shallow, and the pupil widely dilated and fixed. An important, if not the fundamental, factor in sympathetic irritation is a reflex hyperæmia; in the severer forms of sympathetic disease there is in all cases, probably, some alteration in the constitution of the intra-ocular fluid, and frequently a copious outpouring from the ciliary processes and the posterior surface of the iris of a highly albuminous coagulable exudation. Such changes, even when of very moderate intensity, are of a kind well calculated to excite an attack of glaucoma in an eye which has any liability to that disorder.

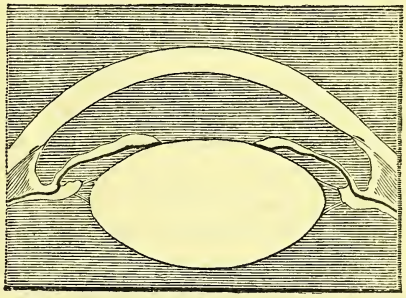

Fig. 139.

From a Specimen of Secondary Glaucoma induced by Serous Iritis.

Showing angle of anterior chamber much distended; tissue lining angle of chamber altered in appearance by fibrinous deposits, which probably obstruct access to Schlemm's canal. (*Enlarged 3½ diameter.*)

The structural changes which have been mentioned in the foregoing pages as commonly accompanying the glaucomatous process, are those which appear to be concerned in the *causation* of increased tension. In addition to these, there are usually to be found in eyes blinded by glaucoma a variety of atrophic and degenerative changes which must be

regarded as *consequences* of the disease. Some of these have been very fully investigated and described by W. A. Brailey.* In very many of the specimens microscopically examined by him, Dr. Brailey found a great enlargement of the calibre of the circulus arteriosus iridis major, together with, in most cases, a thinning of the arterial wall; and he found, in all but very recent cases, an atrophic condition of the ciliary muscle, as evidenced by a greater or less reduction in its bulk, and more or less sclerosis of its fibres. These changes were, on the whole, most pronounced in specimens of primary glaucoma. They bore a close relation the one to the other, though the arterial appeared sometimes to have preceded the muscular in point of time. Although a different significance has been given to them by their discoverer, there is the strongest reason, I think, for regarding them as *results* of the obstructive condition. When the excretion of the intra-ocular fluid is checked, the secretion thereof must be checked also; and when the outlet is completely closed the flow of fluid from the ciliary processes must cease. It is just in this advanced stage—the stagnation stage—that the arterial dilatation and the accompanying muscular atrophy are most pronounced; in specimens of very recent glaucoma they are either wanting or very slightly marked. Dilatations are often observed in vessels the arterioles or capillaries of which are destroyed or in any way obstructed. In the cases now in question the processes are not only subjected to a passive hyperæmia and a diminished possibility of discharging their secretion; but they are also directly compressed or squeezed between the advancing suspensory ligament and lens on the one hand, and the ciliary muscle, iris, and cornea on the other. This state of things may well explain the dilatation of the arteries and the atrophy of the ciliary muscle, and may account for the reduction in the thickness of the latter being sometimes much greater at one portion of the circle than at another. Dr. Brailey records some striking instances in which the arterial and muscular changes were confined to that portion of the circle where closure of the angle of the anterior chamber had occurred—in one case apparently with eccentric displacement of the lens in the same direction.

One other fact must be noted here—a fact which appears at first sight to disprove the essential importance of the obstructive changes which have been described. Complete closure of the angle of the anterior chamber is sometimes present in eyes which are not glaucomatous—nay more, in eyes the tension of which is below the normal standard. In every such instance, I believe, other morbid conditions may be discovered which will explain the non-occurrence of glaucoma. It will be found, namely, either that the secretion of the intra-ocular fluid has ceased by reason of disorganisation of the ciliary processes, as in specimens of old irido-cyclitis, with shrunken vitreous, totally detached retina, and minus

* W. A. Brailey, M.D., "On the Pathology of Increased Tension of the Globe," "R. L. O. H. Rep." IX., p. 199. "A Further Contribution," &c., IX., p. 379. "A Theory of Glaucoma," X., p. 10.

tension; or that the intra-ocular fluid has found an exit through some abnormal channel, in sufficient quantity to neutralise the effect of the obstruction of the normal channel, as in specimens of anterior staphyloma, in which the true corneal tissue has been replaced by a thin permeable pseudo-cornea thrown out by the anterior surface of the iris. I have elsewhere figured and described specimens and cases illustrative of this matter.*

This section of our subject may conclude, then, with the following general statement. The researches of the past few years have shown that glaucoma is the outcome of an obstruction in the path of the intra-ocular fluid. The obstruction is different in different forms of the disease. Its situation is usually such as to conceal it from inspection in the living eye, but its nature may commonly be inferred from the visible conditions. Thus, when, as in primary glaucoma, the pupil is patent and the anterior chamber shallower, or at least not deeper, than in health, we may diagnose a displacement forwards of the base of the iris caused by the pressure of the ciliary processes. When fluid is retained in the posterior aqueous chamber, bulging the iris forwards in advance of the lens, the obstruction must be looked for at the pupil. In both cases the displacement of the iris leads to a closure of the angle of the anterior chamber, which locks up the whole eyeball so far as the intra-ocular fluid is concerned. When the surplus fluid collects in the anterior chamber, and displaces the iris and lens backwards, then the cause of the impediment must lie in the anterior chamber itself, and is to be sought either in the constitution of the aqueous humour, or in the tissues in the region of the ligamentum pectinatum and Schlemm's canal. Hyperæmia and hypersecretion act as exciting or auxiliary causes of glaucoma when conditions which predispose to obstruction are present; they do so by causing a rapid development of the obstructive changes. Hence, though glaucoma is essentially a local malady depending upon structural alteration in the eye itself, it is frequently associated in its outbreak with general or remote disorders which, through nervous and vascular disturbance, affect the ocular circulation.

SYMPTOMS OF GLAUCOMA.

The symptoms of glaucoma are numerous and somewhat complex, and they vary greatly in different forms of the disease, but their comprehension presents but little difficulty when they are referred to their common cause, the excess of pressure within the eye.

An increased tension of the eye is the leading and essential symptom. The ability to detect this change should, therefore, be assiduously cultivated (p. 42) by all who may have to deal with ocular disorders. The examination is made as follows:—The patient is told to close the eyes gently, as if asleep; forcible closure of the lids raises the tension of the eye,

* "Glaucoma: its Causes, etc." pp. 194, 195.

and renders the lids rigid and thereby vitiates the examination. The surgeon, steadying his hands by resting the other fingers on the patient's forehead, places the tips of the two index fingers upon the upper eyelid, through which he can feel the upper part of the globe behind the corneal region. One finger steadies the eye by pressing against it with a suitable degree of force, while the other estimates the tension. The pressure of the fingers should alternate somewhat as in palpation of an abscess suspected of containing pus. Examinations made by the pressure of a single finger, or even with two fingers of the same hand, though sometimes practised, are nearly worthless. It is important to note that an eye which to casual inspection presents no sign of mischief, may be found, when tested by the fingers, to be extremely tense, and that if the digital examination be omitted a case of glaucoma may not improbably pass for one of "amaurosis" with the inexperienced observer; and again that a state of things bearing a superficial likeness to an acute "ophthalmia" may depend solely upon glaucomatous tension of the eye, and can by no possibility be relieved until this change has been detected. On the other hand, it must be observed that a sense of fulness within the eye, experienced by the patient, is no proof that an excess of tension actually exists; and, further, that it is an error to suppose that prominence of the eye involves a change in the pressure within the globe—in some instances it does so, but by no means as a rule. The educated touch of the surgeon will readily obviate all errors of this kind. Mechanical tension-measurers, or tonometers, of various forms have been devised and used for the purpose of determining the tension of the eye more accurately than is possible with the fingers; the employment of such instruments, however, requires much practice and expenditure of time, and the results obtainable even with the best of them can lay no claim to mathematical accuracy, so that for most practical purposes the digital method remains the readiest and the best.

In fundamental principle the mechanical is identical with the digital method. Pressure is made upon the eye so as to impress its surface; the relation between the amount of the pressure and the depth of the impression is the measure of the tension. By the digital method these two factors are estimated by the sense of touch alone; by the mechanical they are both—provided the tonometer be worthy of its name—indicated as known amounts. In my previous essay ("Glaucoma: its Causes, etc.," pp. 38–48) I have described and figured a tonometer designed by myself, which has rendered me good service in studying the pathology of glaucoma. Its principle is the following:—The tips of three light ivory rods which protrude from the front of the instrument are brought into contact with the surface of the eye on a horizontal line 4 Mm. below the lower margin of the cornea. Each rod moves independently in the direction of its length, and the movement of each is indicated by a pointer travelling along a scale, the movement being multiplied twenty times. The central rod is acted upon by a weight in such a manner that its tip makes a pressure upon the eye which is equal under all circumstances to 15 grammes. This rod, therefore, makes a small pit or impression in the surface of the eye, which is deeper or shallower according to the state of the tension; the positions which the pointers assume upon the scale indicate the depth of this pit. Thus, the depth of the pit made by a known pressure is the measure of the tension of the tunics, and

indirectly of the intra-ocular pressure. Unfortunately for all methods of ophthalmotonometry which have been hitherto devised, and, in fact, for all which appear to be mechanically practicable, the ratio which the depth of the tonometer-impression bears to the internal pressure of the eye is not constant; it is affected by several factors, the influence of which cannot be accurately ascertained, *e.g.*, the size of the eye, the shape of the eye, and the degree of elasticity possessed by its tunics, From a theoretical point of view, it is worth while to note that if it were possible to measure the *diameter* of the impression instead of its *depth*, we could calculate the internal pressure therefrom. If to the surface of a sphere in which an impression is produced by external pressure we imagine a plane surface to be applied, the line of contact is a circle; let the term *impression-plane* denote the imaginary plane bounded by this circle; then the following rule holds good for all cases, irrespective of the size of the sphere, the amount of elasticity possessed by its walls, and the degree of pressure, internal and external. It assumes, however, a perfect pliability in the walls:—

RULE.—*The external pressure equals the internal pressure per unit of area multiplied by the number of such units in the impression-plane.*

For example, if a weight of twenty pounds applied externally produce an impression-plane equal to twenty square inches, the internal pressure equals one pound per square inch. Surmising the existence of such an equation, and being unable to test it mathematically, I did so in the first place by experiment. My friend, Mr. Mason Worthington, M.A., F.R.A.S., of Clifton College, has since furnished me with a mathematical demonstration of its truth. Even this method, however, would fall short of the desired end, for it would only tell us the amount of the internal pressure during the formation of the impression in the eye, whereas it is the pressure as it exists unmodified by the use of the instrument which we desire to know; moreover, it leaves out of account the amount of resistance due to rigidity in the tunics themselves. It is certain that, however faultless the construction of the tonometer, and however skilful its application, it cannot be made to yield exact indications of the intra-ocular pressure. So long as eyes differ in size, shape, elasticity, and rigidity, exactitude will be unattainable. But since these same sources of error beset the digital method, also, in addition to one or two others peculiar to itself, especially the varying thickness and firmness of the eyelid, it by no means follows that the mechanical method is useless. My own experience is that the tonometer indicates differences of tension smaller than those which can certainly be detected by the finger, its superior delicacy being chiefly conspicuous in consecutive examinations of the same eye under varying conditions. In certain experiments and investigations it is indispensable to employ some method which, whether more accurate or not, shall, as far as possible, eliminate the effect of the investigator's own wish or prejudice; and here the tonometer gives invaluable aid; in practice it is little likely to become a substitute for palpation by the fingers.

The other symptoms of glaucoma are closely related to the increased tension; they vary in character and in intensity according to the rapidity with which the internal pressure rises, the time of its duration, and the height to which it reaches. In accordance with these variations we recognise three forms of primary glaucoma—the chronic, the sub-acute, and the acute. Each possesses distinctive clinical features, but is not divided from the next by any hard and fast line.

CHRONIC GLAUCOMA (Syn. Glaucoma Simplex) begins almost imperceptibly, progresses slowly, with little tendency to exacerbation or remission, and leads inevitably, unless arrested by treatment, to blindness. The patient, when first seen by the surgeon, is *usually* at least fifty years

of age. He complains of a dimness of sight, which has come on gradually in one, or in both eyes. One eye is almost invariably somewhat worse than the other in this respect; and, amongst the poorer classes, relief is frequently unsought until the second eye is seriously impaired; indeed, it occasionally happens with unobservant persons that one eye is blind, or nearly so, before its owner discovers that anything is going wrong. We are frequently told, if the question is asked, that spectacles have been used from an unusually early age, and that recently they have been changed for stronger ones, once or more, with only temporary benefit. In some cases we learn that the dimness of vision when first perceived varied a good deal at different times of day, or from one day to another; that sometimes in broad daylight objects appeared partly veiled in mist, and at night every luminous object was surrounded by a halo or circle of prismatic colours; while at other times these symptoms would pass off again entirely (see sub-acute glaucoma). In many cases, however, we obtain no history of any variations of this kind, and it is impossible to assign any particular time as the commencement of the loss of sight. The patient complains of little or no pain in the eyes themselves, but will perhaps have suffered more or less in the temples, forehead, or face.

Externally the eyes exhibit little or nothing amiss. The anterior ciliary arteries and veins (*cc'*, Fig. 2, p. 8), the latter of which are barely visible in the healthy eye, are usually somewhat enlarged, and appear as four single or double trunks opposite the insertions of the four recti muscles. The depth of the anterior chamber is reduced by an advance of the lens and iris towards the cornea. The sensibility of the cornea is diminished, slightly at first, greatly in the later stages. The pupil is frequently large in proportion to the age of the patient, and responds imperfectly to light; but this is not a constant symptom. The lens may appear, on simple inspection, to be wanting in transparency, an illusion which is due partly to its advanced position, partly to the dilatation of the pupil, and partly to the increased reflex proper to every senile lens (p. 460). The inexperienced observer may well be astonished to find that he can view with the ophthalmoscope the minutest details of the fundus through a crystalline lens, which to the naked eye looks like a cataract almost ready for extraction. At every ophthalmic institution, probably, instances of bitter disappointment due to previous mistakes in diagnosis from this cause are witnessed: patients suffering from advanced glaucoma, but hoping to be cured of what they have been told is cataract, have to be informed that their malady has been mistaken, and is in an incurable stage.

The tension of the eyeball is increased. The excess at first may be so slight that, apart from other symptoms, it would hardly justify a diagnosis; it may nevertheless suffice, if continuous, to induce the whole chain of secondary changes. On the other hand, it may, in the later stages, amount almost to stony hardness, and yet, if its advance have been regular and slow, it may be entirely unaccompanied by pain, and may alter the

appearance of the eye but little. The increased tension is, however, in this as in every other form of glaucoma, the chief diagnostic symptom.

On examining the functions of the eyes, we find the range of accommodation extremely small, if not totally wanting; the refraction, in the majority of cases, hypermetropic; the acuity of vision more or less impaired; and the field of vision peripherally contracted—a sign that the corresponding portion of the retina is no longer sensitive. The last-named symptom is one which should particularly engage the attention of the surgeon, for besides being an important element in diagnosis, it will aid him to estimate the gravity of the case, and the probability of its being benefited by operation. In order to map out the visual field correctly a perimeter (p. 94) is necessary, but, without the use of any instrument, a sufficiently effective examination may be readily made with the hand alone (p. 92). The loss of sight commences at the extreme circumference of the visual field, and is at first discoverable only when the examination is made in a subdued light. It advances steadily from the circumference towards the centre, but not equally in all parts of the circle. The temporal, the upper, and the lower quadrants of the retina usually suffer more than the nasal quadrant; in other words, vision *outwards* remains unimpaired the longest. The contraction continues until only a very small oval or slit-shaped field remains, which corresponds to a minute area of the retina extending from the yellow spot outwards to the disc; ultimately this succumbs also. It is probable that the progressive contraction of the visual field is intimately dependent upon injury done to the nerve fibres in the area of the optic disc (see below). Leber has shown that the peripheral parts of the retina receive the fibres which occupy the centre of the optic disc. The excavation of the disc occurs at the expense firstly, and mostly, of these central fibres, and it is only in cases of cupped disc that we see the gradual slow contraction of the field. In acute glaucoma, in which no cupping is present, there is not any concentric extension of the blindness at all comparable with this; the function of the retina suffers, as a whole, more than in chronic glaucoma. Moreover, I have ascertained by experiment upon myself, by means of an apparatus which enables one to exert a gradually increasing pressure of known amount upon the eye, that while it is easy to diminish or to annihilate the function of the retina as a whole, it is not possible to obtain a gradual centripetal contraction of the field of vision similar to that which occurs in chronic glaucoma.*

Next to the excess of tension, the chief diagnostic symptom is revealed by the ophthalmoscope. The optic disc, and the vessels lying on it, exhibit changes which are quite characteristic of glaucoma. As the result of pressure, and consequent atrophy of tissue, the disc is depressed to a lower level than that of the surrounding retina—it becomes excavated, or "cupped." The rim of the cup is firmer, and, consequently yields less

* "Glaucoma: its Causes, etc." p. 88.

than the other parts, so that it comes gradually to be undermined in such a way that the sides of the cup are hidden from the observer's view by the overhanging margin. The undermining is made evident by the interrupted course of the vessels, which, visible at the bottom of the cup, are lost to view as they ascend the sides, and reappear, changed as to number and position, as they bend round the rim to gain the retina (Fig. 140, also Fig. 6, Pl. v). When the disc is being viewed by the indirect method, the presence of the excavation may be brought out very clearly by making small lateral movements of the object-lens. The vessels which lie in the plane of the retina have a greater apparent movement than those situated at the bottom of the cup, by reason of their smaller distance from the lens, and consequently seem to outrun and travel in front of these latter. In the direct examination the depression is manifested by the fact that the retina and disc have a different refraction—they cannot be viewed simultaneously; thus, if the rim of the cup be emmetropic, the bottom, being more distant, will

Fig. 140.

CUPPED DISC OF GLAUCOMA. RIGHT EYE.

Showing deep excavation; disappearance of vessels behind margin of cup; displacement of vessels towards nasal side; engorgement and slight tortuosity of veins.

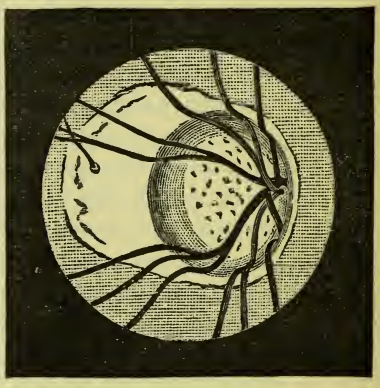

Fig. 141.

CUPPED DISC OF GLAUCOMA ASSOCIATED WITH POSTERIOR STAPHYLOMA. RIGHT EYE.

Showing large size and apparent oval form of cup; absence of abrupt undermining; attenuation of vessels; crescentic atrophy of choroid.

be myopic. From the grade of the myopia—that is, from the strength of the concave lens required in viewing the bottom of the cup—the depth of the depression may be estimated. When a glaucomatous excavation of the disc is associated with staphyloma posticum (that is, with extension and attenuation of the adjacent sclera), its characters differ somewhat from the ordinary. Its diameter is greater; its form appears oval instead of round in consequence of its oblique position in relation to the axis of the eye; its sides have a gentler curvature, and are less abruptly undermined; while the vessels are attenuated by elongation, and are unusually free from sharp bends or curves (Fig. 141). There are two conditions of the optic disc with which the glaucoma-cup may occasionally be

confounded. Firstly, the central depression which is visible in almost every healthy disc may constitute, when larger and deeper than usual, and especially when undermined as it sometimes is, a "physiological cup" which somewhat resembles the excavation which is due to pressure (Fig. 142, also Fig. 2, Pl. I.). The distinction is this: the excavation of glaucoma involves the whole area of the disc, whereas the physiological excavation never does so. Secondly, atrophy of the optic nerve, from intracranial and other causes not connected with any excess of pressure within the eye, leads to loss of substance in the disc, with retraction of its surface (Figs. 4 and 5, Pl. V.). In this latter case the depression is shallow and saucer-like, the edges are never undermined, and there are no breaks or sudden bends in the continuity of the vessels as they leave the disc. Instances of cupping of the disc certainly do occur in which, with a doubtful state of tension, and no clear indication of glaucoma, it is difficult to assign the cause. Possibly the pressure which is proper to the healthy eye may occasionally suffice to excavate a disc of low resisting power; but such instances are very rare in comparison with those in which the diagnosis of a pressure excavation may be made with certainty.

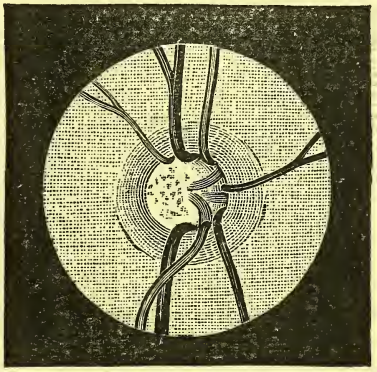

Fig. 142.

"Physiological" Cupping of Healthy Disc. Right Eye.

Showing a large and abrupt natural depression simulating a glaucoma cup, but differing from it in being confined to central portion of disc.

Around the margin of the glaucomatous disc there is almost always a narrow circle of rather irregular contour and of a lighter colour than the adjoining parts—a circumscribed choroidal atrophy.*

The vessels of the disc and retina exhibit changes as regards size, course, and pulsation. The veins frequently give evidence of obstructive engorgement; they are enlarged and tortuous, and in rare instances may even show peculiar bead-like dilatations.† The arteries are usually reduced in size, but not to any great extent in the earlier stages of the disease. The peculiar interruption of course which the blood-vessels appear to undergo at the margin of the disc has already been mentioned. There is frequently another abnormality of distribution—namely, a displacement of all the vascular trunks towards the nasal side of the disc, or, if viewed by the indirect method, an *apparent* outward displacement. Pulsation of one or more of the venous trunks on the disc is commonly to be seen in chronic glaucoma, and, if not present, may be induced by light pressure on the eyeball. It is limited to a small portion of the vein immediately

* Fig. 6, Pl. V.

† Liebreich's "Atlas," Plate XI., Fig. 1.

adjacent to its point of exit, and is not a pulsation in the ordinary sense, that is, not a direct continuation of the arterial blood-wave, but a result of *external* pressure upon the vein, produced by the incoming arterial wave, and transmitted by the contents of the globe. This venous pulsation, or collapse, is of small value, however, as a symptom of increased tension, for it is not present in every case of glaucoma, and it is frequently met with in healthy eyes, especially in those of children. The explanation of this apparent caprice is probably to be found in certain slight differences in the course and arrangement of the veins and their relation to the arteries on and near the disc.* Pulsation of the arteries of the disc is a more trustworthy sign of increased pressure within the eye; but this, too, is occasionally seen in connection with other conditions, and is absent in many glaucomatous eyes.

The whole of these symptoms are not met with in every case of chronic glaucoma, nor are they necessary for the recognition of the disease. By far

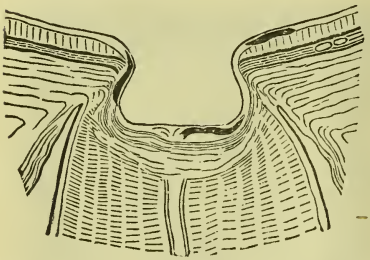

Fig. 143.

LONGITUDINAL SECTION THROUGH OPTIC DISC, FROM SPECIMEN OF CHRONIC GLAUCOMA.

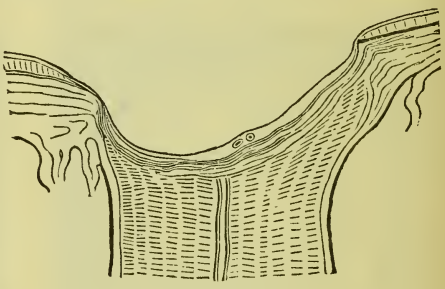

Fig. 144.

LONGITUDINAL SECTION THROUGH OPTIC DISC, FROM SPECIMEN OF GLAUCOMA ASSOCIATED WITH POSTERIOR STAPHYLOMA.

the most significant are the increase of tension, the cupping of the disc, and the contraction of the field of vision.

SUB-ACUTE GLAUCOMA is characterized by the intermittent nature of its course. It may be described as chronic glaucoma, plus periodic exacerbations, of an irritative, quasi-inflammatory type. It is met with as a rule at a somewhat earlier age than the purely chronic, non-irritative form of the disease, but, like the latter, is essentially a malady of the second half of life. Its early or premonitory stage consists of slight occasional glaucomatous attacks, alternating with quiescent periods during which the eye appears to return to a perfectly healthy state.

The chief symptoms in the earlier attacks are a rise of tension remarkable rather for the rapidity of its onset than for its amount, and a peculiar obscuration of the sight. The patient complains that in the day-time he sees all objects as though through a mist or fog, while at night the flame

* "Glaucoma: its Causes, etc." pp. 82–86; also description of Fig. 1, Pl. 1.

of the lamp, or gas, or candle, is surrounded by a circle of prismatic colours. The acuity of vision, tested by Snellen's types in a strong light, is not necessarily reduced below the normal standard in a slight attack, but the letters appear to the patient to be gray rather than black. The coloured zone surrounding luminous objects is strongly indicative of glaucoma. Its characters are as follows:—*The flame is seen with nearly normal clearness; around it is a dark, non-luminous zone, the breadth of which corresponds at all distances to an angle of 4° to 5°; surrounding this is the coloured zone, which has a breadth equal to an angle of 2° to 2.5°, and a total diameter of about 10° to 11°; in the coloured zone the whole of the colours of the spectrum are visible, the violet being invariably on the inner, the red on the outer border; the appearance of the zone is not altered either as to size or as to position of the colours by the use of convex or concave lenses, and it is not altered by variations in the diameter of the pupil; it is perceived not only in direct vision, but also, though with less distinctness, when the image of the flame falls on other parts of the retina than the yellow spot. It is probable that these visual phenomena depend upon slight changes in the cornea, induced by the sudden increase of tension.

The frequency with which these early attacks recur depends upon a variety of accidental circumstances. Hunger, cold, fatigue, respiration of impure air, nausea, fright, anger, sleeplessness, anxiety—in general terms, all conditions which induce *nervous depression*, and which are associated with *dilatation of the pupil*, appear to act as the immediate exciting causes. In this connection it should be noted that an attack may be lighted up in great severity by the local application of atropine to the eye, and has been known to follow the administration of belladonna in other ways. The explanation of this fact is given in the section which deals with treatment (p. 635). The duration of the attacks varies considerably, the earliest being sometimes so transient as to create but little alarm in the patient's mind. The symptoms pass off spontaneously when the patient is refreshed by warmth, food, or sleep, as his condition may require. Sleep especially, even though it be only of five or ten minutes' duration, has in many cases a strikingly beneficial influence; a walk in the sunshine will sometimes cut short an attack; the application of a myotic (eserine, for example) may usually be relied on with confidence to produce the same result. In short, as Laqueur points out, the influences under which these glaucomatous attacks subside are those which, in contrast with the exciting causes above mentioned, induce *contraction of the pupil.*

Although the first few attacks may apparently amount to nothing more than transient obscurations of vision, it is usually not long before more pronounced symptoms are added. Pain is felt in the eye, and often in the

* Laqueur. "A. f. O." 26, II., p. 7. "The Premonitory Stage of Glaucoma: Clinical Study."

forehead and the temple; the conjunctiva is injected; the anterior ciliary vessels are engorged and tortuous; the pupil is unusually large and sluggish, and the anterior chamber shallow; and, on attempting to view the fundus of the eye with the ophthalmoscope during such an attack, we observe some impairment of the normal transparency of the cornea. Each recurrence tends to intensify the mischief and to render the subsequent remission less complete, and by degrees the glaucomatous condition becomes permanently established. The tension remains persistently in excess; excavation of the optic disc, and permanent contraction of the visual field begin. The course of the malady is still marked by periods of exacerbation, between which months may elapse, and during the quiescent intervals the eye may exhibit only such symptoms as are met with in the earliest stages of chronic glaucoma. On the other hand, the attacks may recur from day to day, and rapidly produce destructive changes. The more acute symptoms tend to become persistent; the enlargement and tortuosity of the episcleral vessels increase; the pupil remains large and fixed, in shape frequently oval rather than round; the tissue of the iris atrophies; the disc is deeply excavated with extensive destruction of its nerve fibres; pain is frequent and severe; and sooner or later the day arrives when sight, long since limited to a small central area of the retina, is totally extinguished.

Acute Glaucoma is distinguished from the varieties already described by the suddenness of its onset, the intensity of all its symptoms, and its little tendency to spontaneous remission. While chronic glaucoma is liable to be mistaken for simple atrophy of the optic nerve, acute glaucoma simulates a general inflammation of the eyeball. The resemblance, although only superficial, has led too often to disastrous results. It cannot be too forcibly reiterated that the whole malady depends upon excessive pressure within the eyeball; we have here to deal with no true inflammation, and cannot cure the disease by any antiphlogistic measures; a lowering of the pressure, and that only, can break the chain of evil consequences. The attack is in many cases preceded, or ushered in, by a premonitory stage such as has been described under the heading of sub-acute glaucoma; in other instances it comes on without warning of any kind in an eye which, so far as the patient can tell, has previously been free from all disorder. The premonitory stage is less frequently absent in the younger than in the older class of sufferers. The immediate exciting causes of the outbreak, as in the case of the milder attacks of sub-acute glaucoma, include various forms of nervous excitement and exhaustion, such as grief, anger, anxiety, sleeplessness, etc.; they include, also, all conditions which tend to promote venous engorgement in the head and eye. Many of these are usually associated with more or less dilatation of the pupil; and here again it must be noted that, when the predisposing conditions (pp. 603–4) are present, an acute glaucoma of great severity may be lighted up simply by dilating the pupil with atropine.

The onset of acute glaucoma frequently occurs at night, or in the early morning, while the patient is in bed. The patient states that she—acute glaucoma is somewhat more frequent in women than in men—was seized with pain in the eye, which soon extended to the forehead and temple, or even affected the whole side of the head and face; that this grew more and more severe hour after hour, and seemed almost to blind the eye; that vomiting set in, and that in a few hours she was reduced to a state of utter misery and prostration. When the surgeon is called to the bedside some hours later, the ocular changes are already very striking. The eyelids are somewhat swelled, the conjunctiva reddened, and perhaps œdematous; the scleral vessels are much injected, not only as regards the four groups of episcleral vessels, which are prominent in the less acute forms of the disease, but as a general hyperæmia over the whole surface of the membrane, and especially in the neighbourhood of the cornea. In the most severe cases a protrusion of the eyeball is discoverable. The tears flow profusely, and the eye, though nearly blind, is intolerant of light. The cornea has lost its polish, and "looks like a glass which has been breathed upon," and, provided a high degree of tension has already persisted some time, its sensitiveness to touch is generally diminished or lost. The pupil is widely dilated and totally inactive—a condition which at once indicates that we have before us no ordinary inflammation; it has, moreover, a grayish or greenish hue, instead of the black of the normal eye. Telling the patient to look downwards, and placing the tips of the two forefingers upon the upper eyelid, *we find the eyeball almost stony hard.* The vision of the affected eye is always much impaired. In very acute cases it may, in the course of a few hours, be reduced to the barest perception of light, or it may be totally extinguished; more frequently, a day or two, or more, must pass without relief before this state is reached. The field of vision, tested by the movements of a candle before the eye, or by throwing light upon it from various points by means of the ophthalmoscope, will be found reduced in size. On attempting an ophthalmoscopic examination of the fundus we are baffled, in spite of the dilatation of the pupil, by the imperfect transparency of the cornea. It used to be supposed that the impairment of the reflex from the fundus of the eye was due to turbidity of the aqueous or vitreous humours; but, while it is certainly probable that some serum may exude from the engorged and obstructed capillaries of the ciliary processes, it is indubitable that the loss of transparency lies chiefly in the cornea. If the perfectly fresh excised eye of an animal be squeezed between the finger and thumb the whole cornea is dimmed, and if much force be employed the dimness is so great as almost to completely hide the iris; the instant the pressure is relaxed, perfect transparency returns. This change may be effected as many times in a a minute as the fingers can be made to act; and if the eye be illuminated with the ophthalmoscope meanwhile, an obscuration very much resembling that which is witnessed in acute glaucoma is seen during every compression. Recent observations show, however, that the glaucomatous

opacity is something more than this. Fluid is found to be collected amongst the corneal fibres and beneath the anterior epithelium. It appears that the exalted pressure within the globe resists the nutrient streams which normally percolate the cornea in a centripetal direction to reach the anterior chamber, and thus sets up a sort of œdema in its tissue.* The obscuration in acute glaucoma is not always sufficient to completely hide the details of the fundus. If a view of the optic disc can be obtained, signs of obstruction to the circulation will be discovered; the arteries are small, and almost empty themselves by a regurgitation of blood during each ebb of the arterial wave; the veins are engorged, and their main trunks, close to the centre of the disc, collapse with each arterial pulse. If the fundus can be more thoroughly examined, extravasations of blood situated in the choroid or retina may sometimes be discovered, though they are less frequent during the advance of the disease than subsequent to its relief by iridectomy, when a lowering of the pressure external to the vessels causes them to rupture.

Excavation of the disc is not to be discovered during, or after, a first attack of acute glaucoma, unless the outbreak has been preceded by a chronic excess of pressure of some months' duration. It cannot be looked upon as a purely mechanical result of exalted pressure; it implies atrophic changes, which, though essentially due to pressure, take time for their development. In cases even of very short duration, however, microscopic examinations of the disc in longitudinal section have shown that the first step towards excavation—viz., a retrocession of the lamina cribrosa—has already set in.†

The intensity of the symptoms of acute glaucoma, and the rapidity of their onset, vary considerably. While the milder cases form a connecting link with the sub-acute variety of the disease, the most severe are so sudden and so violent as to well deserve the name *fulminating* applied to them by von Graefe. The stage of total and irremediable blindness is reached far more rapidly in this than in the other forms of the disease.

Absolute Glaucoma (Syn. Glaucoma Consummatum).—The condition of a glaucomatous eye which has already passed unrelieved into total and irremediable blindness is known as absolute glaucoma. The ultimate effects upon the eyeball of an excess of internal pressure are manifested in this stage. They are chiefly alterations in the size and shape of the organ, dependent upon degeneration of its tissues. The symptoms belonging to the absolute stage of glaucoma vary somewhat in accordance with the previous character of the disease. If it have been chronic throughout, the eye, though totally blind, may appear at first glance to be perfectly healthy, and may be entirely free from pain. Its extreme hardness to the touch, however, at once declares the cause of

* Fuchs.—Pflueger. *Vide* "Ophth. Review," Vol. II., pp. 127 and 246.

† W. A. Brailey, "R. L. O. H. Rep." Vol. IX., p. 208.

the blindness, and the ophthalmoscope reveals a deep excavation of the optic nerve, with contracted arteries, and a surrounding ring of choroidal atrophy. The anterior chamber is usually shallow, but the pupil, even in this stage, is not always dilated; it may be of normal size, and retain its power of consensual action. Occasionally the anterior chamber increases in depth, and attains to a greater size than in the healthy eye. Large tortuous blood-vessels may sometimes be seen traversing the surface of the iris. An eye blinded by chronic glaucoma may remain for years without undergoing any visible external change; ultimately, however, it will almost certainly suffer certain of the alterations described below.

When the course of the disease has been sub-acute or acute, the ultimate changes are usually much more prominent. The anterior ciliary vessels are large, tortuous, and of a deep purplish red colour, the large trunks appearing in front of the insertions of the recti muscle, and ramifying and inosculating as they approach the situation where they perforate the sclera. Their appearance is all the more striking because the general surface of the sclera is unduly pale and the conjunctiva thin. The transparency of the cornea is more or less impaired, its epithelium is thickened, rough, and sometimes raised in little vesicles; and the true corneal tissue is often more or less densely opaque, especially throughout that portion exposed by the opening of the eyelids. Its sensibility is greatly lowered or abolished. If the structures behind the cornea are visible, the iris is seen to be reduced to a narrow circle by reason of the wide dilatation of the pupil; its characteristic markings are gone, and at the peripheral line it is in contact with the cornea, its surface being altered in colour by destruction of the epithelium. The lens sooner or later becomes cataractous, the opacity being frequently of that brilliant white or yellowish character which is met with in secondary cataracts associated with advanced disease of the ciliary processes, and, provided the cornea be transparent, it then becomes unpleasantly conspicuous, by reason of its advanced position and the wide dilatation of the pupil. It is most important, however, to remember that, in any stage of glaucoma, the lens may appear at first sight to be cataractous when it is not so, and that this resemblance may lead at an earlier stage of the disease, as already pointed out, to a disastrous error of diagnosis. Pain often continues long after the total extinction of sight. This is so, chiefly, when the disease has previously run a sub-acute or acute course, with sudden painful exacerbations. It is chiefly for the relief of pain that advice is sought when glaucoma is already absolute. Subjective visual sensations are not infrequent long after the eye is blind, and these also occasionally lead the patient to the surgeon, in the vain hope that they indicate a chance of improvement.

The last stage of glaucoma is marked by changes in the size and shape of the eyeball. These occur in two opposite directions, according to whether the continuity of the tunics or the processes of secretion within hold out the longer. If secretion persist in sufficient amount, the eyeball

gradually enlarges, the tunics are thinned, the curvature of the cornea flattens and approximates to that of the sclerotic, the ciliary sclera is extremely attenuated, and has a bluish look in consequence, and the conjunctiva is atrophied and rotten. Instead of a general enlargement, or simultaneously with it, a more circumscribed bulging of the sclera frequently occurs in one or more places. Such a weakened state of the tunics is liable to lead to rupture. This occurs usually during some excessive muscular effort—such as coughing, sneezing, or the like—which is accompanied with forcible contraction of the eyelids; it is followed by profuse hæmorrhage into the eyeball, and generally causes violent pain until the distended tunics are relieved by the free escape of blood. Ultimately the eyeball collapses and contracts to a small size, in which condition it may unfortunately still be liable to recurrences of pain. If, on the other hand, the destruction of the secreting organs have proceeded in advance of the thinning of the sclera, changes of an opposite kind occur —viz., shrinking, with loss of tension. The cornea contracts in all diameters, the sclera is indented by the traction of the tendons of the recti, its resistance to the finger is greatly lessened, and a small, atrophic, sunken eye remains. Even in this condition also there is sometimes a liability to attacks of pain. Inflammation of the choroid, followed by ulceration and rupture of the cornea, extrusion of necrosed tissues, and even hæmorrhage of considerable amount, may complete the destructive process.

Secondary Glaucoma is always a serious complication, whatever be the nature of the pre-existing disease; and, in cases of certain kinds, it is one for which the surgeon should be constantly on the watch. Its occurrence may at any time necessitate a prompt alteration in the line of treatment. For example: a sudden rise of tension during the course of a severe iritis, or kerato-iritis, may demand the performance of an operation which would otherwise be contra-indicated by the inflammatory state; it may, in a case of traumatic cataract, call for the prompt evacuation of lens matter which one would otherwise prefer to leave to spontaneous absorption; it may oblige us at once to suspend the use of atropine, however desirable on other grounds, and to employ eserine in its stead.

An increased tension is the essential and characteristic symptom, as in the case of primary glaucoma, and is commonly the only one of much diagnostic importance. Impairment of vision, pain, and vascular injection, if already present, are immediately aggravated by the onset of the glaucomatous condition. The pupil dilates if it be free to do so—as, for example, in cases of serous iritis and intra-ocular tumour. The field of vision, if it can be tested, and if the retina be not already disorganised, will be found to undergo a progressive centripetal contraction under long continuance of the pressure. The optic disc, if visible, will be seen in course of time to suffer excavation. In short, we may say that the symptoms of secondary glaucoma are the same as those of the primary disease, except in so far as

they are modified or masked by the presence of other morbid changes in the eye.

Changes in the form and size of the globe are especially frequent in secondary glaucoma, because this form of the disease is not confined to the latter part of life, but is often met with in the youthful eye, the envelopes of which are far more readily distensible than those of the adult and senile eye. Buphthalmos (p. 296), is a constant result of long-continued excess of pressure in the eye of the child. And again, the varieties of the staphylomatous condition are far more numerous and irregular in the secondary disease, because of its frequent connection with inflammations and mechanical injuries of the tunics. Thus, localised staphyloma, single or multiple, occurs in the ciliary region or at the equator when an inflammation of the uveal tract, involving also the sclera and retina, is complicated with excess of pressure; and partial or total staphyloma of the cornea, or of the cicatricial tissue which replaces it after ulceration or injury, frequently arises under the same condition. Such prominences are not certain indications of excess of pressure from within, for they may occur merely under the action of the normal pressure upon weakened tunics, but they should always suggest an examination for the presence of morbid tension. The ultimate fate of such eyes, if the glaucomatous condition pass unrelieved, is the same as that to which primary glaucoma leads; namely, either loss of tension with detachment of the retina and shrinking of the whole globe, or progressive distention, attenuation, rupture, and collapse.

TREATMENT OF GLAUCOMA.

In the treatment of glaucoma the surgeon should have one end and aim constantly before him, and one only—the reduction of the increased tension. If this end be thoroughly and permanently attained, the whole train of symptoms will be arrested, and cure, so far as it may be possible, will be achieved. The practical question, therefore, which should at once arise whenever we are confronted with the malady is this:—By what means can the increased tension be reduced most safely and most surely?

Before this principle was realised glaucoma was an incurable disease. Every eye which it attacked was doomed to blindness. Since von Graefe made his beneficent discovery, hundreds of persons every year have been saved from this calamity by the means of cure which he placed at our disposal—the operation of iridectomy.

Of late we have acquired a therapeutic agent (eserine) which is of great value in many cases of glaucoma, and a new operative procedure (sclerotomy) which may in some cases advantageously supersede the older operation; but it is not too much to say that the value of iridectomy still surpasses that of all other measures taken together. Incalculable mischief would be done should the discussion of these newer modes of treatment obscure the fact that iridectomy, as practised by von Graefe, still stands pre-eminent as *the* remedy for glaucoma.

Eserine, the active principle of the Calabar bean, applied in solution to the eye, effects in many cases of glaucoma a well-marked reduction of the tension. Laqueur was the first to publish evidence of this important fact. Adolph Weber observed it, and employed it in practice, independently. The drug is now very widely used in special practice for the purpose named, being of great value as a preliminary and auxiliary to operative treatment, but rarely supplanting it altogether.

In *chronic* glaucoma the usefulness of eserine is usually less conspicuous than in the more acute forms, though even in the former it will sometimes reduce the tension for a while, and thus delay, though it cannot arrest, the progress of the disease.

In *sub-acute* glaucoma it is sometimes of the greatest service. The earlier and milder attacks often yield at once to its application, and thus the disease is kept at bay for a while, and time is gained for the consideration of operative treatment, a respite which is most grateful to the patient, and which may materially facilitate the conduct of the case.

A Case in Point.—A mechanic, aged thirty-eight, whose right eye had been blinded many years previously by an injury, came to me with typical premonitions of glaucoma in the left eye. He still pursued his work, in spite of temporary obscurations of vision occurring almost daily. He was informed explicitly that his sight would certainly be lost sooner or later unless he underwent operation; that "drops" would probably help him for a while, but would not effect a cure; that operation might fail, but in all probability would permanently cure; and that he must very shortly choose between the certainty of ultimate blindness on the one hand, and the slight but immediate risk of operation on the other. The daily use of a drop of eserine solution produced a marked improvement, and for a while he evidently cherished the belief, in which he was strongly upheld by his wife, that he should not need to submit the eye upon which his livelihood depended to the chances of an operation. At the end of two months, however, the prognosis was verified; exacerbations became more frequent and more serious, and yielded less completely to the eserine; and work was no longer possible. Of his own accord he then asked for operation with as little delay as possible, and added that, however it might turn out, he should always know that the right thing had been done. An iridectomy upwards gave an excellent result.

In *acute* glaucoma, also, eserine sometimes does signal service, lowering the tension in a few hours almost to the normal standard, and thereby greatly facilitating the performance of iridectomy should this still be necessary. Its prompt application is especially likely to do good if the attack have been excited by the use of atropine. In certain other cases eserine proves completely powerless; and, in some instances it distinctly aggravates the sufferings of the patient, by the pain which it produces, even if it do not actually intensify the glaucomatous condition.

Unfortunately it is impossible to predict with certainty in any given case whether the use of eserine will be beneficial or not, but we shall certainly gain in confidence and success if we bear clearly in mind what is the change which we hope to effect by the application of the drug. Eserine is not to be regarded as a specific for increased tension in general,

but as a means of combating a particular displacement of the iris, which, in a large class of cases, is the immediate cause of an excess of tension. Eserine is the direct antagonist of atropine. It is only too well established by experience that atropine aggravates primary glaucoma, and may even induce an attack in an eye which previously had shown no symptom of the disease. It does so, probably, by a purely mechanical process. As the pupil dilates, the tissue of the iris is gathered into wavy folds or convolutions which considerably increase the thickness of the membrane (*vide* Fig. 146). If the angle of the anterior chamber be previously as narrow as is compatible with the discharge of its excretory function, this thickening of the iris may easily suffice to embarrass the escape of the intra-ocular fluid, and then with the first rise of tension the ciliary processes swell up and intensify the obstruction, and glaucoma is established (*vide* p. 606). Eserine, on the other hand, by strongly contracting the sphincter of the pupil, stretches out and attenuates the tissue of the iris and thereby tends to reopen the angle of the anterior chamber.

This explanation is based on good evidence. Clinically we see that it is chiefly in the earliest stage of primary glaucoma that eserine is powerful for good, and atropine for evil. In this stage the periphery of the iris is pressed upon behind by the ciliary processes and is in contact, or very nearly in contact, in front with the cornea; this is a condition which contraction of the pupil may relieve, and dilatation aggravate in the way described. In the advanced stage the periphery of the iris is firmly adherent to the opposing surface of the cornea; this is a permanent obstruction to the escape of the intra-ocular fluid which cannot be influenced by contraction or relaxation of the sphincter of the pupil, even supposing that the iris is not already completely paralyzed. It is in the sudden but comparatively mild attacks which come and go at intervals during the premonitory stage of the disease, when the outlet of the chamber appears to be constantly on the brink of danger, but never occluded for very long together, that eserine acts with the greatest promptness and certainty. In very severe attacks of acute glaucoma adhesion of the iris occurs early, sometimes certainly within two or three weeks, and the pupil soon loses its contractility, hence all chance of benefit by eserine soon disappears in such cases.

In the second place, eserine and atropine cause no marked changes of tension in the healthy eye. It is true that eserine increases the flow of the blood to the iris, and probably to the ciliary processes, and that atropine diminishes it in a very marked degree, and there is reason to believe that these vascular changes are accompanied by a corresponding increase and diminution respectively in the interchange of fluids within the eye,* but any variations in the intra-ocular pressure which result therefrom are usually so slight as to be almost indiscoverable. If eserine does alter the tension of the healthy eye it raises it; if atropine affects it, it lowers it. Hence it is clear that the very marked effects of these drugs in glaucoma are not due to any

* Ulrich. "A. f. O." 26, III., p. 35.

influence over the processes of secretion. Figures 145, 146, and 147, which are faithful representations of actual specimens, exhibit the causes of the different effects of atropine and eserine upon the tension, in the normal condition of the eye, in recent glaucoma, and in glaucoma of long duration.

Fig. 145.

From a healthy Eye at Age 65. (*Magnified* 15 *diameters.*)
Showing a condition in which eserine and atropine cause no decided alterations in the tension of the eye.

Fig. 146.

From a Specimen of recent Acute Glaucoma. (*Magnified* 15 *diameters.*)
Showing an obstruction productive of high tension, which may often be relieved by eserine, and aggravated by atropine.

Fig. 147.

From a Specimen of Chronic Glaucoma of Long Duration. (*Magnified* 15 *diameters.*)
Showing an obstruction productive of high tension, which is not influenced by eserine or atropine.

Finally the matter is put beyond dispute when we find that in certain varieties of glaucoma in which the mechanical action of the iris is reversed—certain dislocations of the lens (*vide* p. 611),—the influence of these drugs over the tension of the eye is reversed also.*

According to my own experience the following are the rules which should guide us in the employment of eserine as a remedy for glaucoma. Every case of primary glaucoma should be treated in the first instance tentatively with one or two instillations of eserine. The eye should be re-examined within twenty-four hours, and, if the case be acute and severe, within a much shorter time. If no diminution in the size of the pupil has then occurred it is improbable that any benefit will be gained by the continuance of the drug, but in the absence of urgent symptoms a few additional instillations may be tried. If, on the other hand, the diameter of the pupil is smaller than before we may expect some advantage, even though it be only an increased facility in carrying out the operation of iridectomy or sclerotomy. Should the tension be reduced by eserine nearly or quite to the normal standard, with a corresponding amelioration of the other symptoms, we may safely persevere in its use, and postpone the consideration of an operation, but it is wise in every case to warn the patient that the drops, though likely to relieve for a while, cannot be relied on as a means of permanent cure. If, in spite of a well marked contraction of the pupil, a fair trial of eserine is followed by no manifest improvement in the state of the tension, the acuity of vision, or the area of the visual field, the propriety of an operation must at once be considered. As a preliminary to iridectomy eserine is advantageous in so far as it reduces the tension of the eye, but it has the disadvantage of increasing the hæmorrhage from the iris. After iridectomy, while the anterior chamber is still empty or only partially reformed, eserine is sometimes useful, but it is apt to promote the formation of posterior synechia, and has been known to induce a fresh glaucomatous attack. Seeing that an operation for glaucoma in the one eye is occasionally the exciting cause of an outbreak in the fellow-eye, it is well to use eserine once or more to the latter as a measure of prevention. The strength of the preparation employed, and the frequency of its application, should, in all cases, be the minimum which is sufficient to contract the pupil and to keep it contracted. A solution stronger than two grains to the ounce of water is probably never desirable, and in many cases a much weaker solution is to be preferred. When eserine proves powerless to contract the pupil it will not reduce the tension, nor do good in any way, but, on the contrary, is likely to do harm by promoting hyperæmia, and should not be used further. In case the watery solution proves after a while to be too irritating, the gelatine discs (p. 324) containing eserine, or those containing the extract of Calabar bean, may be substituted. Another myotic, pilocarpine, the active principle of jaborandi, may be used for the same purpose.

* *Vide* "The Action of Atropine and Eserine in Glaucoma." "Ophth. Review," Vol. I., pp. 78 and 113.

Iridectomy is the appropriate treatment for the great majority of cases of glaucoma. The success of the operation in saving or restoring the patient's sight depends almost entirely upon its being performed early in the course of the disease, before the excess of tension has had time to do permanent structural mischief to the retina. Hence the dictum, *When the diagnosis of glaucoma is established, operate.* We have seen that advantage may be gained by preceding the operation by a tentative use of eserine; in a few cases it may be well to substitute a modified operation; in a few others it is proper to abstain from operation altogether; but these reservations hardly diminish the force and value of the above rule. Even now, after iridectomy has been an established remedy for five-and-twenty years, the evil consequences of its omission are far more frequently to be seen than those which follow its injudicious use.

The urgency for immediate operation naturally depends greatly upon the acuteness of the malady. In *acute glaucoma*, unless a well marked improvement is at once obtained by the use of eserine, iridectomy should be performed "without the slightest hesitation or the smallest delay." When the symptoms become intense within a few hours of their onset, every hour which the operation is delayed may induce irretrievable mischief. So long, however, as any perception of light remains, and even although it has already been extinguished for some days, iridectomy should on no account be withheld. The prostrate condition of the patient must not be admitted as a reason for delay. Iridectomy is the surest means of procuring ease and sleep. A delay of some hours is generally inevitable from one cause or another; during the interval a subcutaneous injection of morphia may generally be given with advantage, unless contra-indicated on general grounds, and eserine should be applied locally to the eye. Measures likely to lessen the determination of blood to the eye—*e.g.*, leeches to the temple and hot bottles to the feet—may sometimes be advantageously employed at the same time, but must on no account become an excuse for delaying the operation. An acute attack of great intensity may, in the course of not many hours, reduce vision from its normal acuity to the barest perception of light. Iridectomy promptly performed, will in all probability restore it nearly to its former condition. But just in proportion to the brilliancy of such a result is the danger of delay. In the course of a very few days the blindness may become absolute and permanent.

In *subacute glaucoma*, also, the importance of early operation is great. Each recurrence of the acute symptoms diminishes the degree to which the sight is capable of restoration. When many such recurrences have already happened, and the visual field, *examined during a quiescent interval*, is peripherally contracted, and excavation of the disc is marked, complete restoration will rarely be obtained. But very considerable deterioration of sight *during an exacerbation* does not preclude the recovery of excellent vision after iridectomy. The prognosis must be founded chiefly upon the state of vision immediately before the exacerbation, if this can be ascertained, and upon the duration of the acute attack, as in the case of acute glaucoma.

In *chronic glaucoma*—the simple, non-irritative glaucoma of Continental writers—as in the preceding varieties, iridectomy is the more likely to prove beneficial the earlier it is performed; but even at the best its action is less certain and less strikingly curative in this condition than in the acuter forms. A positive improvement of vision is seldom to be hoped for. Atrophic structural changes here advance *pari passu* with the loss of visual function, and the most that iridectomy can effect is, by removing the active cause, to arrest the further progress of the mischief. Even this result, however, cannot be depended upon in every case. Not unfrequently the effect of the operation proves transient and imperfect, and it occasionally happens that even when skilfully performed it is followed by a painful aggravation of the glaucomatous condition, with speedy loss of whatever vision the eye previously possessed. In every case of chronic glaucoma, therefore, the responsibility of advising operation is a heavy one, and should on no account be undertaken without a full explanation to the patient or his friends of the certainty of blindness on the one hand, and of the uncertainties which beset the operation on the other. If both eyes are decidedly glaucomatous, but both have still a useful amount of sight, iridectomy on one, probably the more advanced, should be performed without delay; the result may decide the treatment of the other. When one eye is already blind, and the vision of the other is restricted to a small central portion of the retina, the chances of retaining useful vision are very small. Having regard to the age of the patient, the impossibility of great benefit, and the possibility of a painful and accelerated progress, a prudent surgeon will only operate on the express desire of the patient to receive the only possible chance of benefit, however small it may be.

Iridectomy, as performed for the relief of glaucoma, consists in the formation of an incision in the sclero-corneal junction, opening the anterior chamber very near to its peripheral angle, together with the excision of the corresponding segment of the iris. Before describing the way in which it should be executed, it will be well to discuss briefly the reasons of its curative power. Much obscurity has surrounded the question in time past, but the difficulty has been in great part removed, I think, by the recent additions to our pathological knowledge.

It may be taken as proved that the tension-lowering power of iridectomy depends essentially upon the incising of the sclera, and not upon the excising of the iris—the success of sclerotomy has taught us this—though it is very far from being proved that the latter step is better omitted. Iridectomy appears to act as follows: in the first place, an immediate lowering of the tension accompanies the escape of fluid from the anterior chamber; this, in the next place relieves the embarrassed circulation, permits the turgid ciliary processes to subside, and thereby tends to reopen the circumlental space and the angle of the anterior chamber; and, lastly, with the healing of the wound, a more or less distensible and permeable cicatrix is formed which acts as a safety valve in the future. The excision of the

iris-segment is necessitated by the tendency which it has, if left, to become incarcerated in the wound, and, moreover, if we succeed in removing it quite up to its insertion we remove the very structure by which the outlet of the chamber is closed during the glaucomatous attack.

Bearing in mind the nature of the obstructive changes which form the starting-point of primary glaucoma (*vide* Figs. 129 and 131), it is obvious that since iridectomy cannot reduce the size of the lens, its efficacy must depend chiefly upon subsidence of the ciliary processes, and should be most conspicuous when the obstruction is due chiefly to their turgescence, namely, in acute glaucoma. This, as we know, it is. It is obvious also that a reopening of the angle of the anterior chamber is far more likely to be attained when the peripheral contact of the iris with the cornea has only recently begun, than when it has existed during many months, and actual adhesion has commenced; this accords completely with our clinical experience that iridectomy reduces the increased tension with greater certainty in the early than in the late stages of the disease.

It appears that, in exceptional cases of very recent and acute glaucoma, the mere evacuation of the anterior chamber by an incision in the cornea may suffice to re-establish the normal tension; the rapid draining away of fluid from the eye, during the few hours which elapse before the incision closes, causing the swelling of the ciliary processes to subside so completely as to restore the eye to its previous condition. But experience has shown that for the great majority of cases a *scleral* incision is imperatively necessary. It permits of a more complete removal of the iris-segment, and the resulting cicatrix appears, unlike cicatrices situated in the cornea, to remain to a certain extent permeable by the intra-ocular fluid. The lips of the scleral incision rarely unite by immediate contact; a grayish semi-translucent cicatricial substance may generally be perceived even where union has occurred most readily, and in many cases this protrudes somewhat above the general curvature of the sclera; frequently, moreover, there is a slight elevation of the adjacent conjunctiva due to the effusion of fluid beneath it. De Wecker relates the case of a glaucomatous patient who, after iridectomy, was in the habit of relieving the slight recurrences to which he was still liable, by making pressure upon the eyeball with his fingers; the effect of this, as witnessed by De Wecker himself, was to extrude a small quantity of fluid beneath the conjunctiva in the neighbourhood of the cicatrix, and to remove at once the glaucomatous symptoms.* Dianoux has recently recommended the systematic daily application of finger-pressure to the eyeball after sclerotomy operations, for the purpose of obtaining a less firm and complete cicatrisation of the wound than would otherwise occur (*vide* p. 648). It is possible, also, that during the healing of the iridectomy incision new vascular channels are formed in the cicatricial tissue and adjacent sclera, which reopen a

* "Thérapeutique Oculaire," Pt. i., p. 381.

passage for the fluid into Schlemm's canal, or the adjoining veins. Whatever be the precise explanation of the permeability, the practical fact remains that the more nearly the incision coincides with the normal outlet — the angle of the anterior chamber — the more certain, *cæteris paribus*, is its efficacy; and this, be it observed, is no theoretical inference, for it was established long before the physiology of this region was understood.

Mode of performing iridectomy for glaucoma.—It is desirable, in most cases, to employ an anæsthetic (pp. 401–6). I have a strong preference for ether, on account of its superior safety. I also habitually administer a draught containing fifteen or twenty grains of chloral, from twenty to thirty minutes previously (p. 403).

The operator stands or sits behind the patient's head (p. 400).

The patient being anæsthetized to complete muscular relaxation, the eyelids are separated with a stop-speculum, the conjunctiva is seized with toothed forceps near to the lower corneal margin, and the eye is rotated downwards, if necessary, so as to render the upper portion of the ciliary region easily accessible. The incision is made by some operators with a broad keratome (Fig. 72, p. 393); by others with a linear knife (p. 395). The linear knife is, in my opinion, to be preferred in every case. Its advantage is that it enables one, without danger to the lens, to effect a longer and more peripheral incision than can be made with the keratome. The latter, if introduced in a plane parallel with the iris, is liable to enter the chamber at some distance from its periphery, even though the external puncture be made well in the sclera; and if a sufficient length of incision is not obtained by the forward movement of the knife, it is extremely difficult to enlarge it satisfactorily during the withdrawal, for the chamber, already abnormally shallow, is instantly diminished almost to abolition, by the forcible escape of the aqueous humour. With the linear knife it is possible, even after the appearance of the point in the anterior chamber, to modify the length and position of the incision according to the space available, by making the counter-puncture a little further forward or backward, as may be necessary. The puncture is made in the sclera at about 1 Mm. from the corneal margin; the point of the knife is carried slowly and steadily across the upper part of the chamber, keeping near to the periphery, and passing very close to the anterior surface of the iris, but without wounding it, until it effects the counter-puncture in the corresponding spot. The distance between puncture and counter-puncture, externally, in a straight line, should be from 8 to 10 Mm. So far the blade of the knife should be kept parallel with the plane of the iris, but as it cuts its way out, and as soon as the angle of the chamber is incised between puncture and counter-puncture, the edge should be turned somewhat forwards, so as to emerge nearer to the corneal margin than it otherwise would. By this means the object desired—namely, an internal incision situated as peripherally as possible is attained, while a large conjunctival flap, which is undesirable, is avoided.

The fixation forceps being entrusted to a skilled assistant, whose duty it is to retain the eye in the necessary position without making any pressure upon it, and with a minimum amount of traction, the iris is seized near to its pupillary border with the small-toothed iris forceps, and drawn out through the lips of the incision; it is then divided by means of fine scissors as close as possible to one angle of the incision (the right), and as nearly as possible down to its insertion; then, by drawing it along towards the other angle of the incision (the left), it is torn from its insertion between these two points; and, finally, it is removed by a second snip with the scissors as close as possible to the latter point. Should a tag of iris remain entangled in either extremity of the wound, it must, if possible, be disengaged and returned into the chamber. This may usually be effected by lightly stroking and pressing on the sclera close to the angles of the incision with a curette or by means of a finger on the upper eyelid; the introduction of instruments into the anterior chamber should as far as possible be avoided. The efficacy of the operation depends greatly upon the thoroughness and cleanness with which the iris segment is removed.

Instead of tearing the iris away, some operators prefer to remove it by a succession of small snips with the scissors as close as possible to the lips of the wound, as by this means the danger of bleeding is diminished. Others, again, content themselves with removing with one cut of the scissors the portion of iris withdrawn with the forceps. The last-named method is by far the easiest of performance, and in some cases of acute glaucoma may perhaps give the desired result, but it is liable to unsatisfactory consequences, and should never be practised.

Accidents of several kinds may occur during and immediately after the performance of iridectomy. Hæmorrhage is inevitable: its importance depends upon its situation and amount. Bleeding from the iris, provided it does not interfere with the precision of the operation, is seldom of any consequence. It is undesirable to attempt to remove all blood from the anterior chamber before closing the eyelids; a little pressure on the outer lip of the incision does all that can be done with advantage or safety. The blood will almost certainly be washed away by the fluid which escapes from the chamber during the following few hours. If the ciliary processes be wounded by the knife, or if, as probably sometimes occurs, they are injured by the tearing of the iris, more serious bleeding will follow. The application of lint, dipped in ice-cold water, over the closed lids for some minutes, and the sparing use of the spatula, so as to separate the lips of the incision, is the best means of treatment. The withdrawal of clots of blood by instruments, or the application of any pressure to the eye is liable to do more harm than it can ever do good. Small extravasations of blood from the retinal capillaries are of very frequent occurrence, as the immediate result of the lowering of the pressure on their external surface. In acute glaucoma they are probably never absent, but, as a rule, have no permanent bad effect. They are always to be found with the ophthalmoscope

in such cases, as soon as an examination of the fundus is possible, and may be entirely reabsorbed during a few weeks. Bleeding from the larger vessels of the retina or choroid is a less frequent occurrence, but much more serious. Occasionally during the operation, more frequently perhaps within the next twelve hours, sudden violent pain is felt in the eye, the tension rapidly rises to more than its former excess, and any sight which existed is totally extinguished. Such symptoms indicate the pouring out of a considerable amount of blood into the posterior part of the eye—between choroid and sclera, between choroid and retina, into the vitreous chamber, or in several of these directions simultaneously. Excision of the eye should generally be undertaken without delay.

Wounding of the lens by the knife-point is a very serious accident. It not only creates a cataract; it aggravates the glaucomatous condition by causing the lens to swell, and leads almost inevitably to loss of the eye. Rupture of the capsule of the lens, especially in the equatorial region, or of its suspensory ligament, may occur independently of any touch of the knife, by reason of the sudden forcible advance of the lens during the escape of the aqueous humour. With due care in operating, neither mishap is likely to occur.

The greater the reduction of the tension immediately after the operation, the more favourable, as a rule, is the prognosis concerning the permanent abolition of the glaucomatous condition.

The after-treatment is simple. A *light* pad and bandage are applied; or the eyelids are merely closed with strips of black court-plaister and protected with a wide and deep cardboard shade. Unless there is a persistent tendency to sickness, I invariably employ the latter method, being strongly of opinion that any external pressure, even such as may arise from a well-adjusted bandage, during movements of the head upon the pillow, is a source of danger to the glaucomatous eye, especially if its tension have not been very thoroughly reduced by the operation. Moreover, this mode of dressing permits the inspection of the eyelids, and the use of hot or cold applications to them without the slightest movement of the patient. Rest upon the back, with absolute avoidance of all straining movements, is carefully enjoined. The occasional application, over the closed eyelids and forehead, of a few thicknesses of soft flannel wrung out in very hot water, will commonly relieve the pain which immediately follows the operation, or occurs within the next twenty-four hours, unless it depends upon some serious complication such as free hæmorrhage into the vitreous.

Prof. McHardy tells me that he adopts and strongly advocates the following mode of dressing:—Over the closed eyelids a double layer of lint is laid, and kept moistened with cold boracic lotion. The case has the constant attention of a nurse, who is provided with a pad of cotton wool, with which she is instructed to afford the eye *temporary* support should the patient strain, through vomiting, sneezing, coughing, or ejecting excreta.

The period which elapses before the wound closes sufficiently to re-establish the anterior chamber varies greatly. Occasionally, when there is

an immediate and complete reduction of the tension, union occurs almost as rapidly and as naturally as after iridectomy in a non-glaucomatous eye. More commonly it is delayed a day or two. As a rule, the delay is longest in cases of chronic glaucoma, with very shallow chamber, in which an excess of tension is still discoverable immediately after the operation. In such cases three or four days, a week, or even longer, may elapse before the iris is permanently removed from contact with the cornea. A re-opening of the wound, accompanied by a sudden sharp pain in the eye, and a gush of hot water from between the lids and down the nostril, may occur once or more during the first ten days.

It has been already stated that the cicatrix which results from the performance of iridectomy on a glaucomatous eye is frequently somewhat elevated above the surface of the sclera. This is attributable to the effect of a continued excess—though frequently a very slight excess—of pressure from within upon the new elastic material which unites the cut edges. When the bulging becomes considerable, it is described as a *cystoid cicatrix.* So long as it appears to produce no irritation or inconvenience it should not be counteracted in any way, as there is good reason to believe that it is a preservative lesion rather than a disaster. If, however, it is a source of pain and a centre of hyperæmia, it should be treated first by simple puncture with a cutting-needle, repeated from time to time as may be necessary. By this means a lowering of the tension of the eye is obtained, which affords an opportunity for subsidence of the protrusion. Should it obstinately recur, the propriety of a more radical treatment—namely, a second iridectomy or a sclerotomy—must be considered.

Malignant Glaucoma is a term used to indicate the aggravated condition which occasionally follows the performance of iridectomy. Excluding those cases of disaster which are due to a faulty execution of the operation, or to some palpable complication such as escape of vitreous or copious hæmorrhage, this malignant course is limited almost entirely to the chronic form of the disease. Its main characteristics are that the lens remains in close and forcible contact with the iris and cornea, little or no fluid escapes from the wound, while the tension increases rather than diminishes; great injection of the vessels in the ciliary region, and sometimes chemosis, occur at the same time.

If the explanation previously given of the pathology of glaucoma, and of the tension-lowering power of iridectomy, be accepted, it is not difficult to understand the import of these untoward symptoms.

In cases which run a healthy course, the operation effects a drainage of fluid from the vitreous chamber; in the malignant cases, the escape of fluid from the vitreous chamber appears to be even more impossible than it was before. If the conditions of obstruction depend upon an unusually large size of the lens, rather than upon turgescence of the processes; if the anterior chamber be very shallow; and if the vitreous pressure be greatly in excess, we have a state of things which must

almost of necessity be aggravated by the withdrawal of the aqueous fluid. Driven forward by the vitreous, the lens applies itself closely to the cornea and intervening iris; its margin lies to the *outer* side of the internal opening of the wound, and thus blocks the channel through which fluid would otherwise drain away; the vitreous pressure, but little lowered by the escape of the scanty aqueous fluid, soon rises to and exceeds its former height; if the lens have any elasticity remaining, its diameter is still further increased by the excessive compression to which it is subjected between vitreous and cornea; and soon the locked condition of the eyeball is even more complete than before the performance of the operation. This explanation of malignant glaucoma will perhaps be regarded as too entirely hypothetical. In its support I would refer the reader to a description given by Adolph Weber of the conditions observed by him in an eye excised on account of "malignant" progress after iridectomy.* I have myself had the opportunity of dissecting four specimens of primary glaucoma in which iridectomy had failed to reduce the tension. The lens was in contact with, and occluded, the wound in every case; but the evidence is not entirely to the point, for in three of the cases there appeared to have been an injury of the capsule of the lens, with some extrusion of its substance; in the fourth the uninjured lens was applied to the cornea. Weber has actually succeeded in obtaining a return of the lens to its normal position, and a refilling of the anterior chamber, by means of paracentesis of the vitreous chamber combined with simultaneous pressure upon the cornea, and has thereby in more than one instance abolished the condition of malignant glaucoma. The procedure, even when carried out with minute attention to the details given by its author, does not always succeed.

Dr. Weber lays stress upon the importance of close attention to several points of detail in performing this operation; it will therefore be well to translate in full the passage in which he describes it. ("Von Graefe's Archiv.," xxiii., p. 1.)

"The sclera is to be punctured with a double-edged broad needle, at a point 8 to 10 Mm. from the corneal margin, and by preference at the outer side, in the horizontal meridian; and while the scleral wound is made to gape by a quarter-rotation of the instrument on its long axis, pressure is made, by means of the upper lid, upon the cornea, in a direction perpendicular to the plane of the coloboma, in the direction of which the lens is usually protruded most; the pressure is to be slight at first, and gradually increased. The patient should persistently 'fix' the operator during this manœuvre, and thereby enable the latter to judge correctly of the position of the cornea, in spite of its being nearly covered by the upper lid; further fixation with instruments, and the distortion of the form of the globe which is thereby produced, can thus be avoided. The maximum pressure is continued for a minute or a minute and a half, so as to allow time for the re-formation of the aqueous humour; blood is generally mixed with the latter, but disappears in four to six hours—a sign that the filtration channels are reopened. A light pressure bandage and rest in the recumbent position for twenty-four hours completely guarantee the result which has been attained. Chloroform is necessary only when the ciliary region is very sensitive, and then the Graefe cataract spoon may be used conveniently as a means of pressure. I have hitherto employed, in addition, the

* "A. f. O.." 23, p. 84. *Vide* "Glaucoma: its Causes, etc." p. 170.

following local treatment before and after the operation, and I believe that it greatly aids the reduction. One or two days beforehand atropine is applied a few times, so as completely to relax the sphincter of the ciliary muscle (sphincter ciliaris), and, about twenty minutes before operating, one or two drops of a two per cent. eserine solution are applied; the commencement of pain in the brow is the signal to seize the instruments. The eserine treatment is continued for some days, until the margins of the coloboma, which are almost invariably adherent to the cornea, are freed. The tension, which previously was extreme, is normal by the evening dressing. The vitreous opacities, which are always present, disappear more rapidly than after successful iridectomy, probably because, in addition to the arrest of the morbid process, a considerable quantity of turbid vitreous escapes and is replaced by new. The most suitable time for carrying out the operation is between the tenth and twentieth days after the iridectomy, by which time the corneal wound is already sufficiently firm to stand such pressure. Clear signs that an acute attack is imminent would necessitate an earlier interference. I have not as yet ventured on this proceeding while the incision is still open. Neither have I had opportunity to attempt it in cases of luxation of long standing. I would give a warning against any modification of this proceeding; I have amply proved that neither the pressure nor the scleral puncture alone suffices to bring about the replacement."

Iridectomy appears occasionally to act as the exciting cause of an outbreak of glaucoma in the fellow-eye. This sequence, though decidedly exceptional, has been too often observed to be a merely accidental coincidence. Its occurrence is by no means paradoxical, when we consider the surrounding conditions. It may be very safely assumed, in such a case, that the second eye was, previous to the operation on the first, either already glaucomatous or in that condition of danger when any slight cause may light up the malady. Expectation of the operation, the operation itself, the effect of the anæsthetic in some cases, the subsequent anxiety concerning the result, perhaps with unrest and sleeplessness, are all calculated to induce a state of mental excitement, which, as we have seen, may and does induce the rapid development of glaucoma under certain predisposing conditions. Again, we know that irritation of one eye from almost any cause is frequently accompanied by some slight temporary weakness of its fellow. Especially is this the case where there is considerable irritation of nerve fibres. Wounds in the ciliary region are, above all others, apt to excite sympathetic hyperæmia, and, seeing that the iridectomy incision lies in close proximity to the line of danger, it appears not unnatural that it should occasionally exercise such an influence over the circulation or secretion in the fellow-eye as, under strong predisposition, will cause a development of glaucoma. In view of such a possibility, it is prudent to inform the patient, or, by preference, his friends, that an attack of glaucoma in the one eye is frequently associated with a tendency to the same disease in the other. As a measure of precaution, it would probably be well in all cases to contract the pupil of the sound eye by means of a very weak eserine solution (½ grain to an ounce of water) before performing iridectomy on its fellow, and to keep it so contracted for a few days afterwards.

A second iridectomy will occasionally effect a complete and permanent reduction of the tension, when the benefit derived from the first has proved

to be incomplete or transient. It should not be undertaken until ample time has been allowed for the subsidence of the fluctuations between normal and abnormal tension which often occur for a month or two after the performance of iridectomy; nor until a very careful trial of eserine has proved that it has no power to subdue the recurrence of the disease. The second operation is usually performed in a direction opposite to the first. It may be undertaken with reasonable hope of success if the previous failure appears to have been due to a fault in the mode of operating; but when a thoroughly correct iridectomy has not succeeded, much reliance must not be placed upon a repetition of the operation. So far as experience enables us to judge at present, it seems that, in some of these cases at least, sclerotomy offers a better prospect of success than a second iridectomy.

It sometimes happens that the deterioration of vision still proceeds, even after the tension of the eye has been brought well within the normal limits by iridectomy,—that atrophy of the retina advances although the morbid pressure which originated it is removed. In such cases nothing can be gained by further operative treatment.

Sclerotomy is an operative procedure which has been practised rather widely during the last few years as a substitute for iridectomy in the treatment of glaucoma. It consists of an incision more or less resembling that of the older operation, without interference with the iris, and is founded on the conviction that the excision of the iris-segment has no essential part in the tension-lowering power of iridectomy.

In the first attempts in this direction the incision was made with the broad lance-shaped knife, and the operation differed from an iridectomy merely in omitting the excision of the iris. Prolapse of the iris into the wound, either immediately or during the following few days, occurred so frequently as clearly to contra-indicate this mode of operating.

A better method was introduced by De Wecker. Eserine is instilled to contract the pupil. The incision is made either with the ordinary linear cataract knife, or with a special knife—a "sclerotome"—which differs from the cataract knife in having a breadth of from 2 to 4 Mm., and a double cutting edge near the point. In general terms the incision may be described as resembling a scleral incision for the extraction of cataract, the middle third of which is left uncompleted. Puncture and counter-puncture are made in the sclera at a distance of 1 Mm. from the corneal margin, and in such a position that the edge of the knife forms a tangent to the extreme margin of the cornea, or lies somewhat more deeply than this, so as to be hidden to the extent of 1 Mm. within the angle of the chamber. This having been done simply by the thrust of the knife, without any sawing movement, the aqueous humour is allowed gradually to escape by a very slight rotation of the knife. The presence of the blade within the chamber prevents the prolapse of the iris into either of the wounds during this escape. When the aqueous humour has been evacuated as completely as possible, but not before, the knife is very slowly and steadily withdrawn.

Further to guard against prolapse, as well as for its specific anti-glaucomatous action, eserine is thoroughly applied before and after the operation. If the linear knife be used, instead of the somewhat broader sclerotome, the incisions effected by the puncture and counter-puncture are enlarged by an extremely slow and cautious sawing movement during the withdrawal of the knife. The operation is performed either at the upper or the lower margin of the cornea, the knife being carried, in either case, in a direction parallel with the horizontal meridian. De Wecker prefers the lower incision as being easier of execution, and involving no traction upon the eyeball by the fixation forceps. Anæsthesia may be dispensed with except in the case of very restless patients. Eserine is instilled, and, if we follow De Wecker, a well-adjusted compressive bandage is applied immediately after the operation (*vide* p. 419 and p. 643).

By this operation, skilfully performed, it is possible to incise the angle of the anterior chamber to an extent which, in the aggregate, exceeds the length of an iridectomy incision, and to do this without incurring any prolapse of the iris. Prolapse will, however, sometimes occur in spite of the previous use of eserine, and in spite of the utmost care in the performance of the operation. It may occasionally be reduced by means of the spatula or by fine smooth-tipped iris forceps. Should reduction prove impossible, the prolapsed portion is to be excised without enlargement of the wound. The excision even of a very small piece of the prolapsed iris, so as merely to make a small aperture, through which the fluid pent-up within it may escape, is sometimes followed by the immediate return of the remainder of the extruded portion to its normal position within the chamber.

Some operators practise an incision which completely divides the sclera between puncture and counter-puncture, and leaves only a bridge of conjunctiva undivided. It is even asserted that a prolapse of the iris leading to permanent distortion of the pupil and staphyloma at the site of the incision, so far from being deleterious, gives the best results, and should be intentionally aimed at. Few will, as yet, be willing to adopt this method.

In order to prevent a too complete re-union of the divided tissues and so to obtain a permanently satisfactory filtration-scar, Dianoux recommends a systematic manipulation of the eyeball, to be begun on the evening of the day of operation and repeated morning and evening for five or six days afterwards. Placing the tips of the forefingers upon the upper eyelid he makes an alternating pressure upon the globe just as in examining the tension; he instructs the patient in doing the same thing for himself. By this practice he claims to further the objects and increase the success of sclerotomy.*

Statistics more extensive than those which we at present possess, and observations extending over a longer period of time, must be collected

* "Archives d'Ophtalmologie," Sept.-Oct., 1883, p. 404.

before the value of sclerotomy can be fully determined. Already we know that every variety of primary glaucoma which is amenable to iridectomy will, in some instances, yield equally well to sclerotomy; but the relative efficacy and certainty of the two operations are still to a great extent undecided.

De Wecker indicates, as a guide to the choice between the two operations, the more or less perfect reaction of the iris to myotics, complete contraction of the pupil under eserine being necessary for the safe performance of sclerotomy. He enumerates the following as the conditions in which it is to be preferred to iridectomy*:—

1. In all forms of hæmorrhagic glaucoma, and in those suspected of belonging to this category.
2. In all cases of glaucoma congenitale—buphthalmos.
3. In all cases of chronic simple glaucoma.
4. Whenever after an iridectomy the vision has deteriorated, or when the good result of this operation begins after a period to diminish.
5. In combating the prodromata of glaucoma.
6. In all cases of glaucoma absolutum, with complete atrophy of the iris, and attacks of pain.

In acute glaucoma no superiority is claimed for sclerotomy. Here iridectomy yields excellent results; and even though a skilfully performed sclerotomy may attain the same end, the greater difficulty of its execution, especially when the pupil does not respond to eserine, contra-indicates its adoption as a substitute for the older operation.

In hæmorrhagic glaucoma, and in buphthalmos, the secondary glaucoma of children (De Wecker's categories 1 and 2), we know only too well that iridectomy is apt to fail, but we do not know, as yet, that a much better fate commonly attends sclerotomy.

Simple chronic glaucoma, and relapses subsequent to iridectomy, seem to be the forms of the disease in which sclerotomy will find its chief field of usefulness. In chronic glaucoma, especially at an advanced stage, iridectomy frequently fails. Sclerotomy has in a considerable number of cases effectually reduced the tension. Neither operation, however, should be recommended in this condition without much caution and reserve. (*Vide* p. 639). With regard to relapses following iridectomy, it is as yet uncertain whether the site of the coloboma or the opposite side of the chamber is the more favourable for sclerotomy. Success and failure have attended it in each situation. Theoretically the absence of the iris segment appears to favour the execution of the second incision.

In the premonitory stage of glaucoma, sclerotomy is recommended because it does not entail that diminution of visual acuity through mutilation of the pupil which is often observed after iridectomy. The advantage is important, but should receive little consideration until it is certain that sclerotomy is equally trustworthy with iridectomy as a means of permanently restoring the tension of the eye to its normal condition.

* "Internat. Med. Congress Transac." 1881, Vol. III.

In *absolute* glaucoma, pain is the reason for our interference. Sclerotomy, though successful in some cases, affords no certainty of benefit. The same is true of optico-ciliary neurotomy.

Until we can rely with certainty on giving immediate and permanent relief by some simpler measure, absolute glaucoma with persistent pain should, in my opinion, be treated by excision of the eye. These patients are commonly advanced in years, and are often reduced by suffering; an operation by which we can secure a prompt and final cessation of pain, and a speedy convalescence, is, for them, greatly to be preferred to one which, at best, can only preserve a useless organ, and which may cause an aggravation of suffering, and lead to excision of the eyeball after all.

Although the value of sclerotomy as a remedy for glaucoma is not yet a matter of common agreement, the success which it has achieved in skilful hands is amply sufficient to justify its further employment.

Other Substitutes for Iridectomy, consisting in various modifications of paracentesis of the cornea and sclera in the ciliary region, have been proposed and practised by Solomon, Hancock, Pritchard, and others. A certain measure of success, especially in acute glaucoma, is attainable by these measures; but they are distinctly inferior to iridectomy, and have never gained a wide acceptance. The same is true of a proceeding by which Argyll Robertson has attempted the reduction of glaucomatous tension—namely, the removal of a small circular piece of the sclera by means of a miniature trephine. An artificial drainage of the eye has been attempted by De Wecker by the insertion through the tunics of a loop of very fine gold wire. It is probable that the excessive reduction of tension which ensues on this proceeding is due to the lighting up of destructive internal changes rather than to the desired drainage. Total disorganisation of the operated eye, and sympathetic inflammation of the other, have followed its performance. The operation can hardly be justified in any case in which blindness of both eyes is not already complete; and for eyes which are totally blind, but which demand operative treatment for the relief of pain, my own conviction is that excision is more truly beneficial than any other proceeding.

General and Constitutional Treatment avails but little when the glaucomatous condition is firmly established; but there are certain general principles which should be systematically observed, and which, in the premonitory stage and when the disease has been arrested by operation, are of much importance.

All conditions which tend to overfill the blood-vessels of the head and eyes, and all conditions which lead to excessive bodily or mental exhaustion, must be sedulously avoided, and when arising must be counteracted with promptness and energy.

Thus direct irritation of the eyes, such as arises from wind, dust, great heat, excessive light, must be guarded against. The prolonged use of the eyes upon near objects, especially in the stooping posture, and by artificial light, must be forbidden. Habits of life which involve an undue mental

strain must be given up. Late hours, hot rooms, and the over-excitement of much society, especially when they induce restless nights and unrefreshing sleep, are sources of danger if glaucoma threatens, or if it have for the time been subdued by operation. Constipation of the bowels, coldness of the feet, excess in eating, and especially in the use of alcohol, must be habitually avoided. A darkened chamber is by no means desirable in the incipient stages of glaucoma; the dilatation of the pupil which it induces is a distinct source of danger, whereas a walk in the sunshine will sometimes cut short an incipient attack. No preparation of atropine or belladonna in any form must be used by the patient.

In cases, such as are not infrequent among women, where a tendency to glaucoma, or the actual existence of the malady in an incipient form, is associated with much nervous excitability, the use of bromide of potassium and chloral may do good service. Morphia subcutaneously will sometimes produce a distinct remission of the glaucomatous condition. Enemata, hot foot-baths, the artificial leech, may also occasionally give timely aid. All such remedies, however, are, be it remembered, to be used strictly as auxiliaries in the treatment of the malady when its presence is declared, and are on no account to be employed as substitutes for those measures which can immediately reduce the tension of the eye.

Quinine in considerable doses is much employed by continental surgeons in the after-treatment of patients who have undergone iridectomy for glaucoma. It is said to be useful by reason of its power to lower the pressure of the blood.

When the tension of the eyeball has been brought well and permanently within the normal limits, we have no longer to combat a condition of glaucoma. Any further deterioration of vision is due to a progression of the malnutrition and atrophy which the glaucomatous pressure has previously induced. If any remedies can benefit this condition, they will probably be such as give strength and steadiness to the circulation and stimulate nerve nutrition—*e.g.*, digitalis, iron, strychnia, phosphorus, and, possibly, electricity.

The Treatment of Secondary Glaucoma consists in the treatment of the diseases of which the glaucoma is a complication, together with, in certain cases, measures directed specially against the excess of tension. In the employment of the latter, success will depend largely upon a discrimination of the way in which the complication has been brought about. Thus the choice between eserine and atropine must be made in accordance with the particular obstructive change which it is our object to remove. For example, eserine lowers glaucomatous tension only when it depends upon an alteration at the angle of the anterior chamber, which is reducible, more or less, by traction of the iris towards the pupil. The exalted tension of serous iritis, with deep anterior chamber, is aggravated rather than relieved by eserine; atropine, to reduce the inflammation, and paracentesis of the cornea, when necessary, to remove the morbid fluid which cannot escape through the usual channels, are here the proper local

remedies. Again, when a secondary glaucoma follows the total occlusion of the pupil with bulging forward of the iris, both eserine and atropine are useless; the removal of a small portion of the iris, so as to re-establish a passage from the posterior to the anterior chamber, if performed in time, removes the complication at once. It is unnecessary again to enumerate the various forms of secondary glaucoma, or to discuss their treatment apart from that of the diseases with which they are associated. In many cases—such, for example, as the high tension which accompanies the growth of intra-ocular tumours, and that which is set up by copious hæmorrhages in eyes which are already blind—treatment directed specially against the glaucomatous complication is wholly out of place.

It is important to remember that an excess of pressure within the eye is less rapidly destructive of the function of the retina in the youthful than in the senile eye, and that operative interference for its reduction may therefore with propriety be withheld a little longer in the former than in the latter case, especially if we can fairly hope for its spontaneous disappearance in the natural course of the associated disease.

NOTE TO PAGE 606.

HOCQUARD and MASSELON have recently recorded a case bearing closely on this matter (*Archives d'Ophtalmologie*, May-June, 1883, p. 231). A female with congenitally small eyes suffered at thirty-six years of age from double chronic glaucoma. One eye becoming quite blind and painful was excised. It presented "a considerable reduction in all its diameters." "The anterior chamber was much reduced in consequence of the enormous relative size of the lens." "The lens appeared enormous only because the eye which contained it was very small. Measured carefully, its dimensions were identical with those of a normal lens during maximum accommodation." Obstructive changes of the usual kind were found, and had manifestly been caused, so the authors expressly state, by the pressure of the processes against the iris. The pathological explanation given is entirely in accord with what has been said above, except that no importance is attached to what I conceive to have been the essential factor—the disproportion between the lens and its surroundings. The case establishes the important fact that the lens may attain to a normal size, though the dimensions of the globe are subnormal.